VOLUME 18 NUMBER 3

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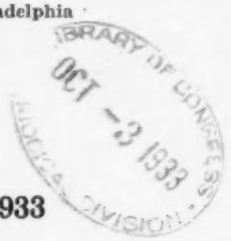

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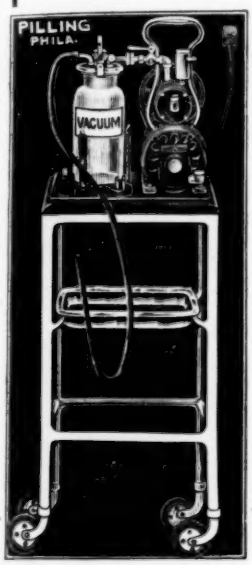

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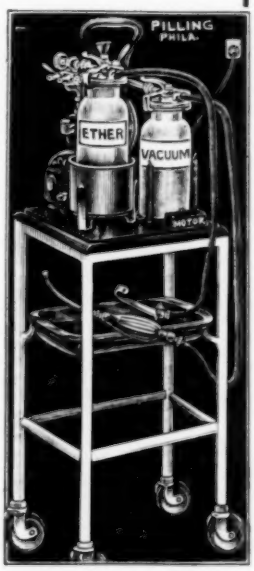

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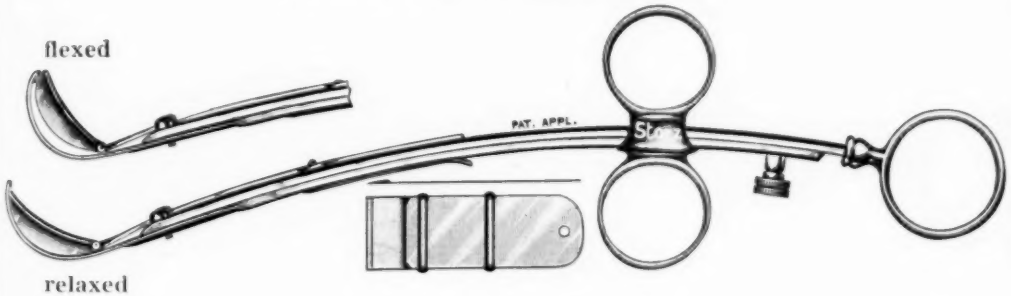

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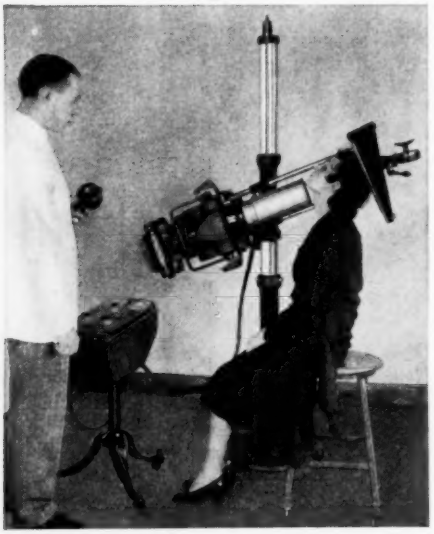

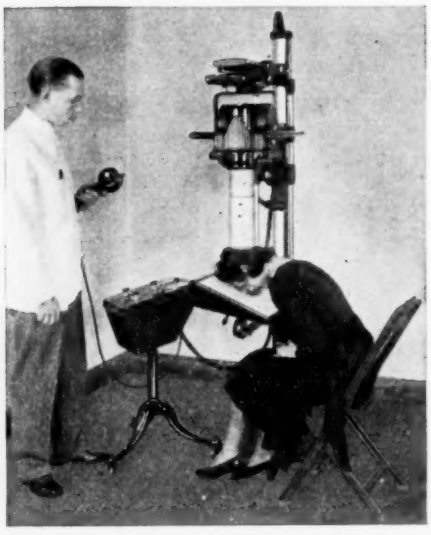

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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 18 SEPTEMBER, 1933 NUMBER 3

NEUROLOGIC PROBLEMS IN OPHTHALMOLOGY AND IN RHINO-OTOLARYNGOLOGY

A. E. BENNETT, M.D.

OMAHA

In presenting so comprehensive a subject as neurologic problems in relation to ophthalmology and rhino-otolaryngology I wish to bring out a few points as they come to the neurologist that seem to indicate the need of a closer cooperation between the neurologist and the ophthalmologist and the rhino-otolaryngologist. I believe that in some instances patients' needs are better cared for by this cooperation, and that at times mistakes in diagnosis or unnecessary delays in adequate therapy are prevented.

NEUROLOGIC PROBLEMS IN OPHTHALMOLOGY

Many neurologic diseases begin with ocular symptoms or, because of the predominance of ocular symptoms, the patient first consults an ophthalmologist. One finds in a few unusual types of conditions that some ophthalmologists do not sufficiently understand the problem to make an early diagnosis.

Oculogyric Crises.—Only in ophthalmologic literature of recent date does one find mention of the frequent disorder of oculogyric crises. This syndrome, in which paroxysmal spasmodic deviations of the eyeballs occur either upward, downward or laterally, is usually a complication of postencephalitic parkinsonism. The first cases that I saw I observed in patients referred to me by the late Dr. Harold Gifford, who thought that they presented hysterical states. Further study of more cases proved that all the patients with this condition had been suffering from chronic encephalitic states. Finally (in 1930) Dr. Patton and I [1] reported a series of cases of this type. Monographs have since been written on the disorder, and large numbers of cases are recognized all over the world.

The symptoms consist entirely of attacks of deviation of the eyeballs upward, to the right or to the left or downward, lasting from several minutes to several days. At times the eyeballs remain fixed in these

Presented before the Omaha-Council Bluffs Ophthalmological and Otolaryngological Society, Feb. 15, 1933.

1. Bennett, A. E., and Patton, J. M.: Oculogyric Crises in Postencephalitic States, Arch. Ophth. **4**:361 (Sept.) 1930.

planes or can be controlled for a moment only to return to the unusual position until the attack has ceased. Fluttering or blinking movements of the eyelids and distressing involuntary closing of the eyelids frequently occur. With the attacks there are often synergistic movements of the head and body. Often an emotional element of fear makes the picture seem hysterical. Careful neurologic study invariably shows the patient to be suffering from a postencephalitic condition and often shows a marked parkinsonian residuum of the disease.

No satisfactory physiologic or pathologic explanation of these peculiar reflex disturbances has been given. Most authorities believe that the origin of the disturbance is in the basal ganglion or the striopallidal system. The condition may be a combination of an emotional state and organic change in the brain, the emotional factors producing a release phenomenon of the controlling, coordinating centers of the eyes. Continuous medication with scopolamine or stramonium is a fairly satisfactory palliative treatment for the disorder. In any unusual spasmodic deviation of the eyeballs one should suspect the possibility of a complication of encephalitis.

Myasthenia Gravis.—This condition is one that frequently is seen first by the ophthalmologist because of the early ocular symptoms, and apparently is frequently not diagnosed at this stage of the disease. The characteristic symptom is muscular fatigability. In the large majority of patients the fatigability shows in the extra-ocular muscles first, beginning with ptosis, diplopia and, at times, facial weakness. At first these symptoms disappear after rest. As a rule the patients awaken in the morning quite normal, but within a few hours the ocular palsies return. The ocular symptoms may predominate for several months before other more characteristic symptoms show themselves. Later bulbar symptoms occur, such as dysphagia, dysmasesia and dysarthria. By this time the general bodily musculature is affected in the phenomena of rapid fatigue. Remissions of the symptoms occur and tend to throw one off guard in making a diagnosis. The disease is frequently seen with exophthalmic goiter. The etiology undoubtedly involves some obscure endocrine metabolic imbalance. The only consistent pathologic finding in the disease is hyperplasia of the thymus. The only abnormal biochemical change found is a disturbance in the excretion of creatine and creatinine, with creatinuria. What the relation of this to muscle metabolism is remains a mystery. There seems to be some imbalance in the amino-acid metabolism.

A fairly satisfactory palliative treatment of this disorder consists in the administration of glycine and ephedrine, which relieves to a large extent the extreme asthenia. The problem of placing patients with this condition under early treatment rests largely with the ophthalmologist. He needs to study any unusual cases of variable, transitory diplopia or recurrent ptosis carefully for evidence of early myasthenic reaction.

Myotonia Atrophica or Myotonic Dystrophy.—While this is presumably a rare disease, yet, like other rare diseases, it is frequently encountered when one is on the look-out for cases. This disease, also, is often seen first by the ophthalmologist, because of the frequency of cataracts.

A few of the outstanding features and their importance to the ophthalmologist seem to me to be worth reviewing. Myotonic dystrophy is a familial disease showing an unusual combination of myotonia (excessive contractility of muscles with lessened power of relaxation) associated with a peculiarly distributed muscular atrophy. In addition to these symptoms there are other widespread dystrophic changes, such as cataract, gonadal atrophy with sterility, baldness and generalized emaciation. The myotonia and atrophy show principally in the muscles of the face, neck and distal extremities.

The frequency of an advanced degree of cataractous change should be of extreme interest to the ophthalmologist. It is probable that all patients with myotonia atrophica show opacities of the lens. There seems to be a characteristic slit lamp picture of the disease that should determine the diagnosis. Dr. Gifford, Dr. Fairchild and I [2] have reported the results of studies made on 4 patients.

Vogt [3] made the most careful study of cataract in this disease. He first described the early changes seen by the illumination of the slit lamp. There is a characteristic occurrence of a mixture of shiny flakes, white dots and fine, dustlike opacities in a sharply defined layer just under the anterior and posterior capsules. These flakes give a play of green and blue characteristic of cholesterol crystals. In about 10 per cent of the reported cases there was an advanced degree of cataract. The numerous extractions of cataracts have given good results. I have seen several cases in which the cataract was removed without the ophthalmologist suspecting the dystrophic background. It would seem that all patients with presenile cataract should have a neurologic and systemic examination in order to determine the etiology. Fleischer,[4] in examining a large group of patients with presenile cataract, found 38 cases of myotonia dystrophia. Apparently the disease is frequently mild and is missed in diagnosis.

Papilledema.—The present criteria for the diagnosis of tumor of the brain must be changed. Earlier diagnosis of cerebral neoplasm is necessary if one is to obtain more satisfactory surgical results. Responsibility for this rests with medical neurologists and with physicians in

2. Gifford, S. R.; Bennett, A. E., and Fairchild, N. M.: Cataract in Myotonic Dystrophy, Arch. Ophth. **1**:335 (March) 1929.

3. Vogt: Klin. Monatsbl. f. Augenh. **72**:421, 1924.

4. Fleischer, B.: Atrophic Myotonia and Cataract, Internat. Ophth. Cong., Heidelberg, 1916; Abstr., Ann. d'ocul. **154**:175, 1917.

other fields, such as ophthalmology, who may see the patients early. Too often recognition of the condition is delayed beyond the time when the best operative results may be obtained. There is a false tradition that the triad of symptoms of intracranial pressure—headache, choked disks and vomiting—is necessary for a diagnosis. It is now recognized that only from 60 to 80 per cent of patients with cerebral tumor have papilledema. Papilledema is more common in association with tumors below the tentorium cerebelli. The large percentage of patients with early tumors in the anterior chamber have no ocular changes. In these patients the use of the spinal manometer establishes the pressure syndrome before fundoscopic examination. Papilledema does not develop as a rule until the spinal pressure is above 20 mm. of mercury. In some cases the spinal pressure remains normal and throws one off guard. When a tumor is suspected and the pressure syndrome is lacking, encephalography finally determines the diagnosis.

Tumors of the Frontal Lobe.—At times a tumor is localized in this area from examination of the eye alone (Kennedy's syndrome). One should remember that a frontal tumor often gives rise to optic atrophy, which is unilateral for a time. Kennedy called attention to the fact that a tumor expanding downward produces retrobulbar optic neuritis, central scotoma and optic atrophy on the side of the tumor and papilledema on the opposite side.

The following case illustrates difficulties in diagnosis that require the combined efforts of the ophthalmologist and neurologist to evaluate the findings properly.

Mr. M. J., aged 39, in February, 1932, was in bed one week with influenza. He never regained his strength, and he noted gradually failing vision. Since July, 1932, he had been unable to read. He stopped work about August. In July Dr. W. H. Stokes found that his vision was 20/40 in both eyes; the visual fields were normal, with a slight vertical nystagmus. By August the vision was reduced to 20/200 in each eye; the fields then showed central scotomas with involvement of the blind spot and a sector defect for color in the lower part of the right nasal field and the lower part of the left temporal field. The findings were in keeping with retrobulbar neuritis.

Neurologic examination at this time gave negative findings throughout. Cisternal puncture revealed normal pressure, and a normal content of cells and protein, but showed a change in the Lange colloidal gold curve of 1122110000. By September the patient had a low grade fever and had mental confusion and lethargy, the vision was reduced to 7/200 and 9/200 and there was definite pallor of the temporal halves of the optic nerve. Neurologic examination still gave essentially negative findings except for lateral nystagmus, and the spinal pressure was again normal, but the protein content was increased. The sinuses were explored, but no positive infection was discovered. The mental changes became progressive.

The general impression of the examiners was that of a toxic or an inflammatory encephalomyelitis or of an atypical multiple sclerosis. At the Mayo Clinic

encephalography revealed a tumor of the left frontal lobe. Exploration disclosed a large inoperable frontal tumor invading the region of the optic chiasm. Death followed.

This case illustrates the necessity of more careful interpretation of the early symptoms and signs. If closer consultation had been carried out between the neurologist and the ophthalmologist the evidence of tumor would have been more striking, and undoubtedly encephalography would have been advised.

Tumors of the Temporal Lobe.—Cushing [5] reported that defects of the perimetric field had been of paramount diagnostic value in localizing 33 of 39 tumors of the temporal lobe. Lillie,[6] in studying 60 patients in whom the presence of tumor of the temporal lobe had been confirmed, found that 43 of 51 showed defects of the perimetric field for either color or form. Eight patients had normal fields. Ten patients had quadrant defects for 1 or more colors, 15 had homonymous quadrant defects for form and colors, 3 had homonymous hemianopia for colors, 15 had complete homonymous defects, and in 9 the fields were unobtainable. In 17 patients the tumors were definitely localized by examination of the perimetric fields alone. Cushing concluded that the ocular syndrome for the early stages of tumors of the temporal lobe consists of normal visual acuity, bilateral choked disks and homonymous quadrant defects either for color or for form.

The possibility of papilledema caused by extradural suppuration, such as suppuration of the sphenoid sinus or blockage of a sinus, is established. When such a diagnosis is definitely arrived at, exploration should be made, provided that no other cause for the papilledema exists.

Retrobulbar Neuritis.—This condition presents a combined neurologic and ophthalmologic problem in which careful studies from both angles often leave the etiology much in doubt. The commoner etiologic factors are: retrobulbar neoplasm, hemorrhage into the nerve sheath, multiple sclerosis, toxemias originating in the sinuses, and circumscribed serous meningitis with or without interstitial nerve change.

The two principal contentions are those with reference to the incidence of multiple sclerosis and invasion from the sphenoid sinus. Some authorities believe that a large percentage of cases are the result of disseminated sclerosis and that sinus disease is rarely a cause.

Ziegler [7] found that 15 per cent of a large series of patients with multiple sclerosis, who were seen in the Mayo Clinic, had retrobulbar

5. Cushing, H.: Distortion of the Visual Fields in Cases of Brain Tumor, Tr. Am. Neurol. A., 1921, p. 374.

6. Lillie, W. I.: Ocular Phenomena Produced by Temporal Lobe Tumors, J. A. M. A. **85**:1465 (Nov. 7) 1925.

7. Ziegler, L. H.: Multiple Sclerosis; Clinical Review and Follow-Up Study, Minnesota Med. **12**:778 (Dec.) 1929.

neuritis with central scotomas at the onset. Langenbeck,[8] in examining 176 patients with retrobulbar neuritis, found that 77 had multiple sclerosis. Vail,[9] on the other hand, believed that sinus infection is a frequent cause, and suggested that the virus of disseminated sclerosis may gain entrance to the central nervous system from the sphenoid sinus in the same way that other organisms producing infectious diseases of the brain have been shown to do. This, he held, is sufficient ground for an early operation, even in cases of multiple sclerosis. The important point is not which school is correct but that the ophthalmologist, rhinologist and neurologist need to work together to make an early diagnosis and to institute therapy that may check the disease.

Both neurologists and ophthalmologists have been lax in not using examinations of the perimetric field frequently enough in suspected intracranial lesions. Routine examination of the fields for form and color should be made in all cases of suspected neoplasm. Eagleton [10a] showed the extreme value of frequent repeated examinations of the field in the localization of abscess of the brain. He found that cerebral edema about the lesion often produced hemianopic indentations of the fields, which were of localizing value.

Migraine.—The problem of hemicrania baffles the neurologist as well as the ophthalmologist and rhinologist. A restatement of some of the phenomena characterizing the attack should not be a waste of time. The attacks are frequently preceded by visual disturbances. There may be spots before the eyes, the visual fields may be contracted, blind spots or scotomas may appear, and even temporary hemianopia or amaurosis may be present. The scintillating scotomas, when present, are characteristic. A point of light appears in the field of vision, generally at one side, gradually enlarges and becomes zigzag, glimmering and colored. All the visual disturbances are fleeting, lasting only a few minutes as an aura to the attack of headache.

Migraine may be associated with other symptoms, such as tremor or convulsions. A special type occurs with ophthalmoplegia. In cases of this type the headache is generally limited to the forehead above the eyes. After the attack one or more ocular muscles, usually the oculomotor, become palsied, with ptosis, strabismus and diplopia. There

8. Langenbeck, K.: Die Neuritis retrobulbaris bei Nerven und Allgemeinerkrankungen, Deutsche Ztschr. f. Nervenh. **1**:235, 1913.

9. Vail, H. H.: Retrobulbar Optic Neuritis Originating in the Nasal Sinuses, Arch. Otolaryng. **13**:846 (June) 1931.

10. Eagleton, W. P.: (*a*) Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the Brain, J. A. M. A. **92**:713 (March 2) 1929; (*b*) Suppurative Meningitis of Otitic and Nasal Origin, Arch. Otolaryng. **15**:885 (June) 1932; (*c*) Intradural Complications of Aural and Nasal Origin, ibid. **4**:69 (July) 1926; (*d*) Unlocking the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex, ibid. **13**:386 (March) 1931.

may also be internal ophthalmoplegia. It may take several weeks for the palsies to disappear, and cases are reported of permanent palsies following several attacks. These cases often suggest an intracranial disease. Occasionally one sees cases of recurrent ophthalmoplegia of the third or seventh nerves occurring without migraine.

NEUROPSYCHIATRIC PROBLEMS IN RHINOLOGY

Sinusitis.—Undoubtedly there is much to learn concerning the nose and the paranasal sinuses as a portal of entry of intracranial toxemia and infection.

The etiologic rôle of sinusitis in abscess of the brain and in septic and serous meningitis is well known. But the relation of chronic sinus infections to more chronic or obscure neurologic and psychiatric disorders is not well understood. When this field is studied I am sure that a great number of facts will be advanced to explain points that are now matters of speculation.

Meningitis.—Suppurative meningitis is undoubtedly more frequently secondary to sinusitis, particularly to sphenoid and ethmoid sinusitis, than has been supposed. Eagleton [10b] found that pneumococcic meningitis, usually of type III, is often secondary to sphenoid disease.

Acute and chronic infections of the sphenoid and ethmoid sinuses at times give symptoms suggestive of serious intracranial disease, but produce only serous meningitis, with increased intracranial pressure and palsies of the cranial nerves, usually of the third and sixth nerves, due to chiasmal cisternal pressure. The symptoms promptly disappear on adequate drainage of the sinus.

The following case is a good example of hyperplastic ethmoid and sphenoid sinusitis producing intracranial pressure and oculomotor paralysis.

Mrs. F. C. S., aged 40, had suffered from frequent colds in the head in the past. The present illness started with an ordinary headache, but within twenty-four hours the patient was unable to leave her bed on account of a bursting headache, roaring in the head, vomiting and intermittent fever. Examination, made ten days after the onset, showed a fever of 103 F., a slow pulse rate, soreness of the entire cranium, with pain on the slightest movement, stiffness of the neck, ptosis on the left side and slight proptosis. Spinal puncture revealed a pressure of 20 mm. of mercury, 4 cells per cubic millimeter and a protein content of 20 mg. per hundred cubic centimeters. A small drainage of fluid relieved the headache markedly. White blood cells numbered 5,500, with 48 per cent polymorphonuclears. Roentgen findings showed increased density through the left ethmoid cells; both antrums were cloudy, suggesting thickened membrane. On exploration of the sinuses Dr. W. A. Cassidy found extensive hyperplastic exudate in the ethmoid and sphenoid sinuses. Within a few days the patient recovered completely and returned home.

Aseptic Purulent Meningitis.—Dr. G. A. Young and I[11] reported 6 cases of purulent meningitis of the benign type in patients who recovered. Four of these were secondary to ruptured sterile abscesses of the brain. In two cases the apparent etiology concerned a subdural abscess secondary to ethmoiditis, and in one, an abscess of the frontal lobe secondary to frontal sinusitis. In one case the abscess followed trauma of the head. This series of cases impressed on us the fact that at times a benign aseptic type of meningitis follows chronic sinus infection. Studies of the spinal fluid give the diagnostic and prognostic criteria, mainly the complete sterility of the fluid. The history of sinus disease, with roentgen findings, determines the etiologic diagnosis.

Relationship of Chronic Sinusitis to Psychiatry.—I should like to call attention to the frequency of sinus operations on psychoneurotic patients. Records at hand show a very high incidence of sinus operations and tonsillectomies on patients whose symptoms are entirely psychogenic. Such operations discredit the rhino-otolaryngologist, just as oophorectomies and appendectomies on patients complaining of chronic abdominal discomfort discredit the abdominal surgeon, and never cure psychoneuroses, even though they may appear at the time to have relieved the symptoms. The patients invariably relapse and later on blame the surgeon. The same statements apply to sinus operations for chronic headache, tic douloureux and other disorders that are of doubtful intranasal origin.

Recent improvement in roentgenographic technic, particularly the use of iodized poppy seed oil 40 per cent, is helping to make diagnoses more accurate. In grave psychoses psychiatrists have not paid sufficient attention to the sinuses as foci of infection. For some time neurologists have been studying cases of this type, particularly mental states of confusion, depression or paranoia, for evidence of intranasal pathologic factors. Every now and then they obtain a prompt gratifying recovery by successful treatment of chronic hyperplastic or suppurative sinus disease.

In England, Pickworth[12] and others studied large numbers of psychotic patients for evidence of sinus disease. Pickworth found 69 cases of mental disorder in which sphenoid sinusitis was demonstrated clinically, and he showed microbic invasion through the bone covering the sinus into the pituitary and cerebral substance. It appears from these studies that one must look to the sphenoid sinuses particularly to explain some of these cases.

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11. Young, G. A., and Bennett, A. E.: Aseptic Purulent Meningitis, Am. J. M. Sc. **183**:550 (April) 1932.

12. Pickworth, F. A.: Perforation of the Pituitary Fossa by a Septic Lesion of Sphenoidal Sinus Associated with Recent Epilepsy and Insanity, J. Laryng. & Otol. **43**:186 (March) 1928.

Eagleton [10e] cited several cases of abscess of the pituitary gland deriving from sphenoid disease. There are probably other infections of milder degree invading the central nervous system from the sphenoid sinuses, e. g., diabetes insipidus. One case of this type is reported in which relief was obtained by sphenoid drainage.

In clinical and anatomic studies of sinusitis in mental disease made since 1924, Graves and Pickworth [13] found that 50 per cent of the patients in a hospital for mental defectives had acute or chronic sinus infection. They believed that diseased sinus membranes, particularly those of the sphenoid sinuses, allow toxic or microbic invasion by way of the blood stream into the brain, especially in the hypothalamic region, which governs the vegetative and metabolic functions of the body. Many psychiatric symptoms suggest imbalance in these functions. At least the studies of Pickworth and Graves indicate that in obscure mental states one needs to consider sinus infection as an etiologic factor more carefully than has been done in the past.

NEUROPSYCHIATRIC PROBLEMS IN LARYNGOLOGY

While decrying the removal of tonsils in functional disorders that are purely psychogenic, neurologists do not hesitate to recommend removal of the tonsils in obscure nervous disorders when a definite focus of infection is found. One still sees tonsillectomies frequently performed in an attempt to relieve the syndrome called globus hystericus. This functional symptom is always a part of the picture of an anxiety hysteria, a psychogenic disease resulting from frustrated emotional outlet, with sexual conflicts, and responding adequately only to psychotherapy. Patients with this condition frequently consult the laryngologist first. I repeat that operative procedures on such patients discredit the medical profession.

The frequency of functional aphonia needs only to be mentioned. This condition is a conversion symptom of the psychogenic disease that baffles all neurologists, namely, hysteria. The only scientific treatment is psychotherapy.

NEUROPSYCHIATRIC PROBLEMS IN OTOLOGY

Vertigo.—The combined efforts of the neurologist and the otologist are often required to establish the etiology of vertigo. Space does not permit much discussion of this symptom. Any condition of true objective vertigo should be studied extensively in order that it may be classified in its proper group: psychogenic, toxic, ocular, peripheral neuritic or involving the central vestibular pathway.

13. Graves, T. C., and Pickworth, F. A.: Sinusitis in the Etiology of Mental Disorders **21**:45, 1928; Pickworth, F. A.: Confusional Insanity with Empyema of the Sphenoidal Sinus, Brit. M. J. **1**:721 (April 20) 1929.

Deafness.—I wish to emphasize the emotional handicap that develops in certain persons, particularly in those with otosclerosis. These persons are often very sensitive and become introspective, depressed or paranoidal, and the psychiatric difficulty may be more disabling than the loss of hearing. They need psychotherapy in order to learn to tolerate their handicap and to overcome their sensitiveness.

Suppurative Intracranial Disease of Aural Origin.—The neurologist sees the end-results, the tragic complications, of aural disease. He can often be of assistance to the otologist in interpreting the intracranial symptoms or signs and thus help to localize the infection and guide the prophylactic or therapeutic management of the case, often to a successful termination. This is another field where close cooperation between the neurologist and the otologist is essential to the patient's best interest. The incidence of intracranial complications in disease of the middle ear and mastoid is very high.

Warren [14] claimed that 1 of 4 patients with chronic aural infection has intracranial complications, and 1 of every 9 with acute otitis has intracranial disease. In the otolaryngologic clinic of the University of Bordeaux [15] from 1925 to 1928 there were 283 patients on whom mastoidectomy was performed; 14 had meningitis, 9 had abscess of the brain, 17 had sinus phlebitis, 3 had septicemia and 2 had abscess of the frontal lobe which had not been recognized.

The malignancy of these complications should keep neurologists and otologists mindful of the necessity for early radical and preventive treatment. Especially is this true of meningitis, as otitic meningitis of the septic type has a mortality of at least 95 per cent; there is no adequate therapy for this condition. Experimental work that is somewhat encouraging is going on, and an occasional cure is reported after the application of certain radical, heroic measures, but neurologists and otologists must increase their efforts to understand the portals of entry of the infection and to institute early, prompt drainage to prevent the spread either by the blood stream or by direct extension into the subarachnoid spaces.

Otologists must now accept the clinical entity of petrositis and suspect the petrous pyramid in chronic mastoid infection persisting after operation. A challenge is now before them to perfect a method of drainage of this area in order to prevent many of the complications from cerebral suppuration.

Eagleton [10d] recorded 3 recoveries in 4 cases of petrous abscesses, which were effected by unlocking the petrous pyramid. Continued sepsis

14. Warren, W. C., Jr.: Intracranial Complications in Purulent Otitis Media, Arch. Surg. **18**:1552 (April) 1929.

15. Faucher, H.: Abscess of the Brain of Otitic and Sinusal Origin, Rev. de laryng. **50**:403, 1929.

with aural discharge after mastoidectomy, with possibly a Gradenigo syndrome and with pain radiating along the fifth nerve to the face, teeth or eye should cause one to suspect the ear as the site of infection. Improved roentgen technic would be a great help in the diagnosis.

During the past year I examined post mortem 3 patients with otitic meningitis who showed petrous abscesses as the foci of infection. In 1 case the diagnosis was made ante mortem, and a surgical approach was attempted.

The differential diagnosis in early circumscribed purulent meningitis, abscess of the brain, serous meningitis or thrombophlebitis is not always easy. To some otologists the neurologist may seem out of place in helping in thrombophlebitis of the lateral sinus. To other otologists I am sure that what help he can give is welcome and is appreciated. The frequent use of the Queckenstedt or the so-called Ayer-Tobey test in spinal or cisternal punctures to determine the presence of subarachnoid blockage gives neurologists an opportunity to aid in the diagnosis of thrombosis of the lateral sinus. This sign, when properly elicited and interpreted, is of great value. The delayed response or the total absence of a rise in spinal pressure on compression of the jugular vein is not pathognomonic of sinus thrombosis, but is valuable confirmatory evidence when accompanied by clinical signs and evidence of local sinus involvement. In double mastoiditis with signs of septic thrombophlebitis it may be the sole deciding factor as to which side is involved in the thrombosis. One should remember that occasionally anomalies in the venous sinuses, with one side smaller than the other, give false positive results in this test. Sinus blockage is determined by a careful comparison of the pressure readings after compression of each jugular vein when the patient is well relaxed. The test is made with the patient lying on the right side, then sitting in the upright position, then lying on the left side. A persistent absence of rise in the spinal fluid pressure of one side usually means sinus blockage. In about a dozen cases studied so far I have found the test indicating the involved side.

Recently I [16] have been observing with cisternal or lumbar puncture manometric phenomena that seem to indicate partial and total blockage of the foramen magnum. Instead of an increase in spinal fluid pressure on compressing the jugular vein on each side there is a fall in pressure, and simultaneous compression of both jugular veins gives either a slight delayed rise in pressure or no change. After release of pressure there occurs a delayed secondary rise in pressure. I have called this a bizarre or reversed Queckenstedt's sign. These phenomena have been observed in a number of patients who had partial or complete herniation of the

16. Bennett, A. E.: Cerebellar Herniation into Foramen Magnum, J. A. M. A. **100**:1922 (June 17) 1933.

cerebellum into the foramen magnum. In partial blockage there seems to be a ball-valve action that produces a drop in pressure on jugular compression and often a secondary rise on release of compression to a higher reading than the initial reading. In complete block there is no change in the pressure reading on compressing the jugular veins. If this sign is confirmed in more cases it will be of value in indicating danger from cerebellar pressure cone that produces medullary paralysis. It should serve as a warning in meningitis and other states of intracranial pressure, such as cerebral edema, tumor and similar conditions, to use vigorous methods of dehydration, ventricular drainage or even cerebellar decompression to prevent the inevitable medullary paralysis. Further study of this phenomenon will establish its value definitely.

I know of no more dramatic results in surgery than are obtained by the early ligation of the jugular vein and the packing of the sinus following correct diagnosis in these cases. It seems to me that the surgeon needs to take a radical, fearless and positive stand in regard to this infection. It is better to make an occasional mistake than to wait too long when treating septic infections of this type.

CONCLUSIONS

If this paper has successfully conveyed one idea, namely, the need for closer cooperation and study between neuropsychiatrists and ophthalmo-rhino-otolaryngologists, it has been worth preparing.

In order to get the greatest pleasure out of the practice of medicine one must avoid the tendency to become a mere technician and strive to make more accurate etiologic diagnosis and to administer early prophylactic therapy. In regard to one's therapeutic mistakes, one should render scientific self-criticism in order to prevent making the same mistake twice.

CYSTS OF THE LARYNX

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Cysts of the larynx occur less frequently than the literature of thirty or forty years ago would indicate. There is no doubt that polypi of various kinds were included when cysts were considered at that time. One can appreciate this, for even at the present time the classification of benign neoplasms of the larynx is not well understood. Cysts do, however, occur more frequently than textbooks and current literature would lead one to believe.

The first laryngeal cyst on record was encountered by Verneuil [1] in the cadaver of an infant in 1852. Durham [2] was probably the first to observe a cyst in a living person. In 1863, he operated on a large cyst which sprang from the laryngeal surface of the epiglottis in a boy 11 years of age. Gibb [3] removed a cystic growth from the vocal cord of a patient in the same year. Of 693 laryngeal tumors which Beschorner [4] reviewed in 1877, 45 were cystic tumors. The first large series of instances of laryngeal cyst, 117 in number, were collected by Moure [5] in 1881. Ullrich [6] included 156 cases in his paper a few years later. Since then there have been several fine articles on this subject, notably those of Salomon,[7] Glas,[8] Schneider,[9] Marx [10] and New.[11] Instructive case reports and short articles have been written by several laryngologists. Some have seen many cases, while others have seen only a few.

From the service of Dr. Sidney Yankauer, Mount Sinai Hospital.

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1. Verneuil: Bull. Soc. anat. de Paris, 1852, p. 103; 1853, p. 8.

2. Durham, quoted by Gibb.[3a]

3. Gibb, G. D.: (*a*) Throat and Windpipe, ed. 2, London, J. Churchill & Sons, 1864, p. 76; (*b*) Diseases of the Throat, ed. 2, London, J. Churchill & Sons, 1863, p. 154.

4. Beschorner: Ueber Epiglottis-Cysten, Berl. klin. Wchnschr. **14**:620, 1877.

5. Moure, E. J.: Étude sur les kystes du larynx, Paris, Delahaye et Cie, 1881.

6. Ullrich, quoted by Marx: Ueber Kehlkopfcysten, Inaug. Dissert., Würzburg, 1887.

7. Salomon, Walter: Ueber Larynxcysten, Ztschr. f. Ohrenh. **62**:49, 1910.

8. Glas, Emil: Ueber Larynxzysten, Arch. f. Laryng. u. Rhin. **19**:285, 1907.

9. Schneider, P.: Zur Genese der kongenitalen Larynxcysten, Ztschr. f. Ohrenh. **64**:358, 1912.

10. Marx, H.: Angeborene Kehlkopfcysten, Ztschr. f. Hals- Nasen- u. Ohrenh. **21**:376 (May 10) 1928.

11. New, G. B.: Cysts of the Larynx, Journal-Lancet **37**:99 (Feb. 15) 1917.

Cysts of the larynx are of four types: retention, embryonal or congenital, lymph or blood and traumatic or implantation cysts.

A great majority of the cysts which have been reported were retention cysts. This type is formed by the closing off of the duct of a gland so that there is obstruction to the outflow of the glandular contents. A retaining sac is thus created which forms the cyst. It is the same mechanism which is responsible for the production of salivary and sebaceous cysts. Most retention cysts are to be found in the region of the epiglottis.

Congenital or embryonal cysts are distinguished by the fact that they contain both endodermal and mesodermal structures. When, however, there is no intermixture of the products of these embryonal layers, it is difficult, in fact, impossible, to distinguish between a retention and a congenital cyst. A combination of the products of the various germinal layers is seen in the tumor laterally placed in the neck, the so-called mixed or teratoid tumor. The first embryonal cyst of the larynx was recorded by Louys.[12] He removed the cyst by means of thyrotomy after a preliminary tracheotomy. At operation the cyst, which appeared to be situated on the right false cord and in the right ventricle, was found to extend upward to the right greater cornu of the hyoid bone, with the periosteum of which it was found to be firmly united. The outer wall of the cyst contained aggregations of lymph cells and islands of cartilage.

It is claimed by Kahler [13] and others that cystic swellings of the epiglottis are due to faulty union of the bilateral anlage of the epiglottis at the point of coalescence of its two segments. Glas [8] believes that cysts form where the columnar epithelium of the respiratory tract meets the squamous epithelium of the digestive tract. New considers this theory as untenable because the epithelium of both comes from the endoderm. He agrees with Schneider that the embryonic cyst is very likely caused by a pinching off of the appendix of the ventricle. The location, form, size and epithelial lining do not assist in the determination that a cyst is of congenital or embryonal origin. When examination does not reveal mesodermal structure, the possibility of a retention cyst must be considered.

The lymph or blood cyst is formed by a localized collection of lymph or blood in the subepithelial tissues. This is caused by a coalescence of lymph spaces in some cases. This type of cyst is extremely rare.

The traumatic or implantation cyst is of relatively infrequent occurrence. It is caused by the implantation of epithelial tissue into the

12. Louys, quoted by Salomon.[7]

13. Kahler, Otto: Ein Beitrag zur Genese der Epiglottiszysten, Arch. f. Laryng. u. Rhin. **17**:8, 1905.

submucous region. Glas [8] and Dean and Gregg [14] encountered such a cyst. The cyst seen by Dean and Gregg was located in the interarytenoid region. It appeared several weeks after the curettage of a tuberculous larynx.

It is difficult to estimate the relative frequency of the various types of cysts because of the difficulty of differentiating between the embryonal and retention types. Cysts may have a lining of squamous, cuboidal or columnar epithelium. If the cyst is tense and lasts long, the pressure will flatten the epithelium or even cause it to disappear, as in one of New's cases.

Age, physical characteristics, symptoms, diagnosis and treatment are important.

The cysts occur at all periods from birth to old age. Oppikofer [15] reported a cyst of the epiglottis in a patient 89 years of age. The cysts are round or ovoid and may be sessile or pedunculated in their attachment; most of them are sessile. The size varies from that of a pinhead to that of a large hen's egg. Very large cysts are not frequently seen, but those reported by New and Shousboe [16] were unusual for their size. Shousboe's cyst measured 6 by 3 cm. and filled the left pyriform fossa so as to immobilize that side of the larynx. The smaller cysts are most frequently found on the vocal cords, while the larger ones are most often attached to the epiglottis. They may be very small and cause no symptoms, but they may grow rapidly so that an acute condition or a foreign body is suspected.

The cysts have thin or thick walls; they are soft or firm, fluctuant or solid, and are frequently streaked with small blood vessels. They contain a fluid that may be thin and serous, or thick, gelatinous or colloidal. Blood has been found in some of them. They are colorless or translucent, grayish, yellowish, pinkish or light reddish.

The relative frequency of the various locations of laryngeal cysts has been given as follows:

	Moure [5]	Ullrich [6]
Epiglottis	50	60
Vocal cords	45	50
Ventricle	8	8
Arytenoid	4	8
Aryepiglottic fold	4	
Ventricular band	2	
Not indicated	4	

14. Dean, L. W., and Gregg, J. B.: Cyst of the Larynx, Laryngoscope **28**:74 (Feb.) 1918.

15. Oppikofer, quoted by Denker and Kahler: Cysten des Kehlkopfes, Handbuch der Hals- Nasen- Ohren- Heilkunde, Berlin, Julius Springer, 1926-1928, vol. 5, p. 393.

16. Shousboe, H. I.: Ein Fall von sehr grosser Larynxzyste, Acta oto-laryng. **1**:408, 1918-1919.

The frequency of the locations of epiglottic cysts, according to Mishkin,[17] is as follows: lingual aspect, 49; laryngeal aspect, 10, and rim, 17.

The traumatic cyst reported by Dean and Gregg appears to be the only cyst that was located in the interarytenoid region. Fourteen of Beschorner's series of 45 laryngeal cysts originated from the epiglottis. A majority spring from the anterior aspect of the epiglottis, while a few originate from the laryngeal side and a still smaller number from the rim of this structure. The vocal cords are next in the order of frequency as the site of origin of these cysts.

SYMPTOMATOLOGY

The symptoms caused by a cyst of the larynx bear a direct relation to the location and size of the offending tumor. Hoarseness or change of voice occurs when the perfect approximation of the vocal cords is interfered with. Varying degrees of hoarseness will result according to whether the cyst is located on the vocal cord, on the ventricular band, in the ventricle or on the aryepiglottic fold. Interference with respiration has been encountered, ranging from mild dyspnea to extreme asphyxia. The dyspnea is in direct proportion to the amount of airway which is occupied by the growth. Inspiratory stridor may be a prominent symptom in the obstructive cases. Dysphagia is occasionally seen when the cyst obstructs the introitus of the hypopharynx. One of New's epiglottic cysts filled the approach to the hypopharynx so completely that fluid was regurgitated through the nose. In addition, adults complain of a sensation of a foreign body. This sensation accompanies more especially the epiglottic cysts.

DIAGNOSIS

The cystic appearance of the new growth and its location, fluctuancy, translucency, size, shape and color will assist in establishing the diagnosis of a cyst of the larynx. This condition should not be confounded with that of a prolapse of the ventricle in which the prolapsed tissue can be replaced, as cysts cannot be forced out of view. In infants an erroneous diagnosis of enlarged thymus or congenital stridor is frequently made; in older children, the diagnosis of papilloma, diphtheria, asthma or foreign body. Of course, examination of the larynx by whichever means is dictated by the age of the patient will readily establish the diagnosis.

PROGNOSIS

The prognosis is grave in the very young. Because of the small size of their structures and the delicacy of their tissues, the management of

17. Mishkin: Ueber Epiglottiszysten, Inaug. Dissert., Königsberg, 1912, p. 808.

these growths in infants is extremely difficult. Many cases have occurred in which it was learned only after the death of an infant by asphyxiation that the child had a cyst of the larynx. Reports of infants found dead under such circumstances have been made as follows:

Author	Age of Infant
Stoerck [18]	5 days
Salomon [7]	28 days
Edis [19]	2 days
Porak and Theuveny [20]	24 hours
Punder [21]	2½ months
Esch [22]	Less than 1 month
Rosenberger [23]	6 weeks

The prognosis may be grave in older patients when the cyst suddenly increases in size. Davis [24] saved the life of a patient who was being asphyxiated by rupturing the cyst with his finger. In a great majority of older children and adults the prognosis would appear to be good. There have been, however, several cases in which adults were asphyxiated before they could be helped. Laryngeal obstruction from cysts, as from any cause, is always a serious condition.

TREATMENT

The treatment varies according to the size and location of the cyst. The age of the patient must also be considered. Simple incision or cautery puncture does not seem to be of value. The growth recurred even after Beck [25] burned a hole through the wall of a cyst in a 5 year old child. In the case of a young infant it would appear that the simplest and best temporary measure is the production of a hole in the wall of the cyst by the cautery. This keeps the cyst more or less empty until such a time as the infant is able to stand its removal. Total removal is the only certain means of cure. The method by which this is accomplished depends a great deal on the location of the cyst and its size.

18. Stoerck, Oskar: Ueber einen Fall kongenitaler Larynxstenose, Frankfurt. Ztschr. f. Path. **19**:149, 1916.

19. Edis, A. W.: Cyst in the Larynx of an Infant, Tr. Obst. Soc. London **18**:2, 1876.

20. Porak and Theuveny: Kyste du larynx, Compt. rend. Soc. d'obst., de gynéc. et de pédiat. de Paris **4**:150, 1902.

21. Punder, Ann: Larynxstenose durch angeborene Kehlkopfzyste, Jahrb. f. Kinderh. **87**:65, 1918.

22. Esch, A.: Zysten in den ersten Lebensmonaten, Internat. Zentralbl. f. Ohrenh. **18**:1, 1920.

23. Rosenberger, H. C.: Cyst of Larynx and Lobus Azygos of Lung, Laryngoscope **37**:360 (May) 1927.

24. Davis, E. D. D.: Cysts of the Larynx, J. Laryng. & Otol. **38**:473 (Sept.) 1923.

25. Beck, J. C.: Cyst of Epiglottis, Laryngoscope **38**:473 (Sept.) 1923.

New used a snare to remove the cyst in his cases, and cauterized the stump which remained with the cautery tip by the indirect method. Thyrotomy has been used extensively for removal of cysts of the larynx. Imperatori [26] employed this type of exposure in the removal of a multilocular cyst of the aryepiglottic region. Suspension laryngoscopy has been used to good advantage in the total extirpation of these growths. Chamberlin,[27] Freudenthal [28] and others have used this method. Some have been able to remove cysts by means of punch forceps. Pharyngotomy has been employed by some when it was felt that the growth could not be removed through the mouth.

REPORT OF CASES

CASE 1.—J.,[29] aged 2 days, was admitted to the hospital because of audible respiratory stridor, transient cyanosis and difficulty in swallowing since birth. Direct laryngoscopy revealed a reddish translucent mass involving the right aryepiglottic fold and extending in front of the same side of the epiglottis. The larynx was markedly displaced to the left. Incision into the wall of the cyst caused the escape of a thick glairy mucus, as a result of which the cyst became smaller. After this the baby cried more lustily. The inspiratory stridor returned in forty-eight hours and continued for seventeen days. The infant died of bronchopneumonia at the age of 20 days. Postmortem examination of the larynx was reported as follows: "In the region of the right pyriform sinus and effacing the right aryepiglottic fold, extending upward to the lateral portion of the right vallecula and downward to about the middle of the inferior cornu of the thyroid cartilage, there is a globular fluctuant mass the size of a large pea, about 1 cm. in diameter. It has a glistening white smooth surface, and displaces the larynx to the left, at the same time occluding the lumen of the oropharynx anteriorly. The epiglottis is not involved in the mass. The glottis cannot be seen from above and behind.

CASE 2.—M. K., a girl, aged 3 years, was brought into the hospital in extreme dyspnea and asphyxia. Tracheotomy was performed at once. She had had a harsh inspiratory stridor since birth and had been seen by many physicians. She was treated at various times for enlarged thymus, asthma, congenital laryngeal stridor, diphtheria and papilloma. Two weeks before admission her condition became worse. There was an increase in the dyspnea, and the stridor was more marked. A nonproductive cough which caused vomiting had persisted until the time of admission. During the past three days the condition of the child had become alarming. Retraction of the chest in every direction was noted. Direct laryngoscopy revealed a large ovoid mass attached to the left aryepiglottic fold, filling the brim of the larynx and encroaching on the glottis. Tracheotomy was performed with the bronchoscope in place. Three weeks later, under chloroform narcosis induced through the tracheal tube by Dr. Branower, a suspension laryngoscopy was performed, and the ovoid mass was excised with scissors and knife. There was a

26. Imperatori, C. J.: Branchiogenetic Cyst of the Larynx Removed by Thyrotomy, Laryngoscope **39**:679 (Oct.) 1929.

27. Chamberlin, W. B.: Cyst of the Larynx, Laryngoscope **27**:622, 1917.

28. Freudenthal, W.: Cysts of the Larynx Removed by Suspension Laryngoscopy, New York M. J. **11**:1080 (June 19) 1920.

29. This patient was seen and treated by Dr. Kramer.

severe reaction twenty-four hours later; the temperature rose to 105 F., and the respirations were 50 to the minute. This reaction soon subsided, and except for some difficulty in swallowing liquids the convalescence was uneventful. A week after operation direct laryngoscopy revealed considerable loose slough in the left aryepiglottic region. Two weeks after operation the wound was observed to be healthy and granulating. The child was discharged as fully recovered one month after operation.

CASE 3.—A boy, aged 3 years, was admitted to the hospital on Sept. 22, 1932. He had had stridulous breathing and had been hoarse since birth. His breathing became more embarrassed following an attack of whooping cough a year before, and his stridor had increased during the past two months. Tracheotomy was performed. One week later direct laryngoscopy revealed a globular swelling occupying the infraglottic region of the left side. It appeared to be in close relation to the inferior surface of the vocal cord and to the vocal process of the arytenoid cartilage. The globular swelling was removed with punch forceps. The tissue submitted for microscopic study was reported as a cyst wall containing columnar epithelium.

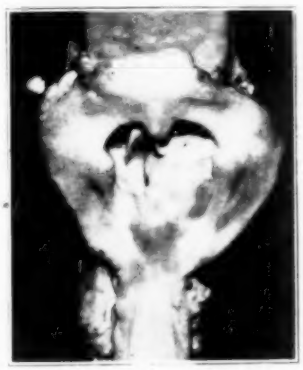

Fig. 1 (case 1).—Autopsy specimen showing cyst of the right aryepiglottic fold which caused obstruction of the pharynx and larynx in an infant, 20 days old.

Although subsequent direct examination of the larynx proved that the cyst was no longer present, the tracheal tube could not be removed. Bronchoscopy revealed a cystic swelling of the right wall of the upper part of the trachea. Thyrotomy and fissure of the upper part of the trachea were therefore performed on November 14. A cystic fusiform swelling was found extending along the right side from the inferior border of the cricoid cartilage to about the third or fourth tracheal ring. The globular swelling previously noted was no longer present. The tracheal swelling was aspirated, but no fluid was obtained. The cystic mass was excised and submitted to the laboratory. The pathologist reported that the specimen consisted of a cyst wall lined by columnar epithelium, infiltrated with lymphocytes and containing mucous glands. The tracheal tube was removed on December 28. There was no hoarseness, and the child was breathing well. About six weeks after he was discharged he began to have some difficulty in breathing. His mother thought that he had a "cold," and paid no particular attention to his dyspnea until it became alarming a few days later. He was seen by a laryngologist, who advised that he be sent to the hospital. The family delayed until the following day. He was then rushed to a local hospital, where he died before I could perform a

Fig. 2 (case 2).—Roentgenogram showing the shadow of a cyst of the left aryepiglottic fold in a child aged 3 years.

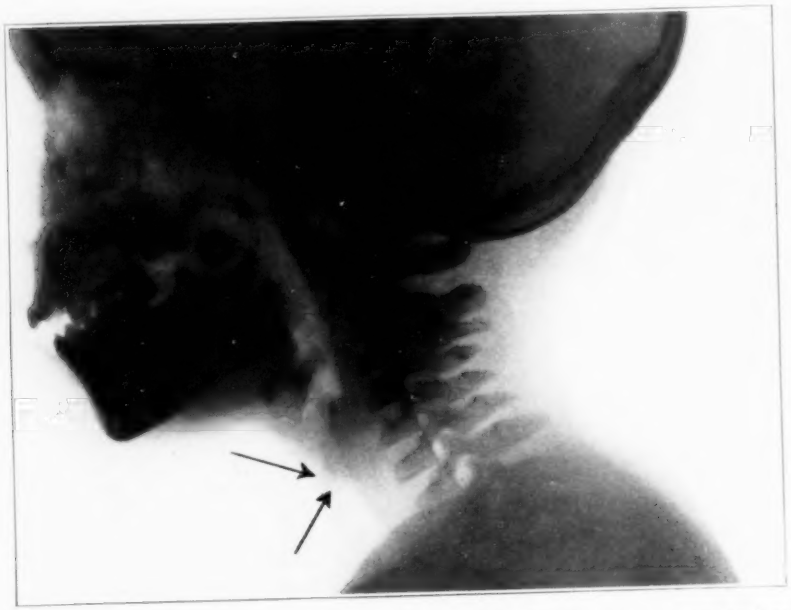

Fig. 3 (case 3).—Roentgenogram showing the subglottic shadow of a tracheal cyst.

bronchoscopy or tracheotomy. A bronchoscope was passed, and artificial respiration with the bronchoscope in place was of no avail. On opening the trachea I was surprised to find a tumor lying unattached in the trachea. A description of the tumor by Dr. Antopol, the examining pathologist, follows:

The specimen consisted of an irregular ovoid mass, 8 by 8 by 6 mm. The surface was smooth except for the region of the pedicle, which was about 1 mm. in diameter. The cut section was edematous in appearance and disclosed numerous

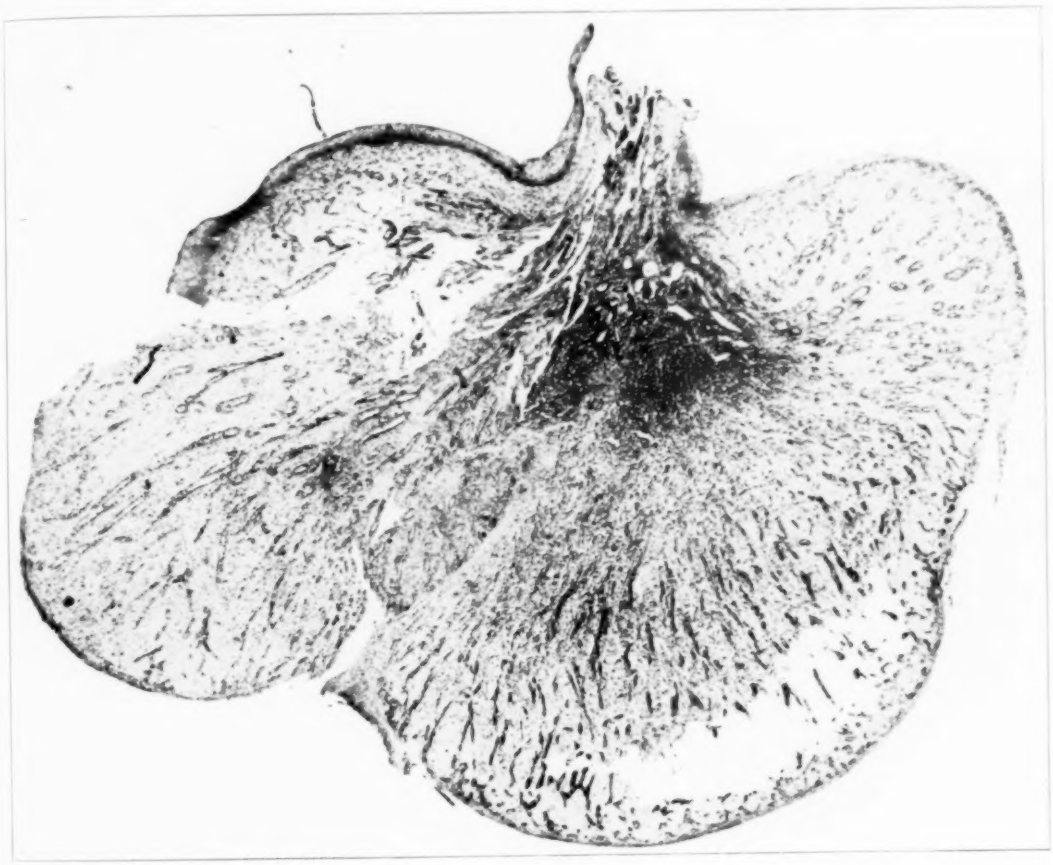

Fig. 4 (case 3).—Photomicrograph showing vascular and edematous granuloma; × 10.

small vessels coursing radially toward the surface. Microscopic study revealed a markedly edematous tissue, the edema being most pronounced at the periphery. Epithelium was not seen on most of the specimen except near the pedicle, where a stratified squamous epithelium with slight cornification was seen. This faded gradually into an edematous and necrotic type of tissue. There was a diffuse round cell and plasma cell infiltration which was most pronounced in the edematous area. Throughout the mass were seen blood channels which converged toward the pedicle. At this point, instead of the edematous stroma of the periphery, there was a denser stroma made up of the fibroplastic type of cells. Van Gieson's stain showed

numerous strands of connective tissue which were separated from each other in the peripheral portion and became much closer at the pedicle. The impression received was that of vascular and edematous granuloma.

COMMENT

It is reasonable to deduce from the history and circumstances of each case that the cyst had been present since birth. In each case only an inferential diagnosis was possible until a direct examination of the larynx was carried out. The third case has several unusual features. The presence of 2 cystic tumors, 1 of which was situated entirely in the trachea, is unusual. The second cyst, which was removed by fissure of the larynx and upper part of the trachea, and the asphyxiation of the child from what proved to be a pedunculated growth, which no doubt sprang from the anterior commissure of the larynx or from the line of fissure of the cricoid or upper part of the trachea, are unusual. The granulomatous and vascular nature of the growth would indicate that it was of relatively recent origin. This tumor mass was of the same type as that frequently seen in adults following thyrotomy. The histologic study of this mass is given in detail because thyrotomy is performed infrequently in young children and because it is thought that the age and delicacy of the structures may influence the type of granuloma which follows this operation. The relatively small size of a child's air passages should lead one to anticipate the formation of a tumor following thyrotomy. It would then be possible to forestall discomfort, distress or death from encroachment on the airway.

Cysts obstructing the laryngeal passage probably account for some of the cases of asphyxia of the new-born. Recently within a period of two weeks a local pathologist encountered 2 such cysts at autopsy, in infants 1 day and 2 weeks old respectively. The first case would have been pronounced one of asphyxia neonatorum if it had not been for the autopsy. It is a fact that this condition is overlooked, not only in the new-born, but also in older infants and young children.

SUMMARY

This paper represents a review of the subject of cysts of the larynx and stresses some of the practical considerations. Three cases are recorded in one of which there were cystic masses in both the larynx and the trachea.

136 East Sixty-Fourth Street.

FORM AND STRUCTURE OF AN AREA OF OTITIC SCLEROSIS IN THE TEMPORAL BONE OF AN ADULT

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AND
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The several histologic features which conjointly characterize otitic sclerosis, or "otosclerosis," are now well recognized, and their occurrence has been described in a recent and detailed résumé of the pertinent otologic literature.[1] The association of the following features, which individually are not limited to bone called sclerotic, constitutes a disease which is seemingly peculiar to the osseous labyrinth: The diseased bone stains intensely; it is richly vascularized by relatively large thin-walled vessels lodged in wide and freely communicating spaces; the spaces are enclosed by definite lamellar systems; the diseased area is quite sharply delimited (by the features just mentioned) from the normal bone in which it is embedded; although bearing some resemblance to a neoplastic formation, the mass is but mildly invasive, not necessarily deforming and usually restricted to certain portions of the capsule; it is not accompanied by an inflammatory reaction.

These, as well as other familiar, but less arresting, qualities were possessed by the specimen which we shall describe. It is our special concern in this paper to discuss the shape of the sclerotic nodule as a whole, and its relation to immediately surrounding portions of the labyrinth.

MATERIAL

The specimen from the collection of the Department of Otolaryngology was obtained from the ear of a man. The block was fixed and stored in a dilute solution of formaldehyde and decalcified in a 3 per cent solution of nitric acid in dilute formaldehyde. Celloidin sections were cut 30 microns in thickness, and stained in either hematoxylin and eosin or van Gieson's stain. The plane of section passed through the sclerotic nodule at a right angle to its long axis, and,

From the Departments of Otolaryngology and Anatomy, Northwestern University Medical School.

Contribution 185 from the Anatomical Laboratory, Northwestern University Medical School.

1. Duel, A. B.: Otosclerosis, New York, Paul B. Hoeber, Inc., 1929.

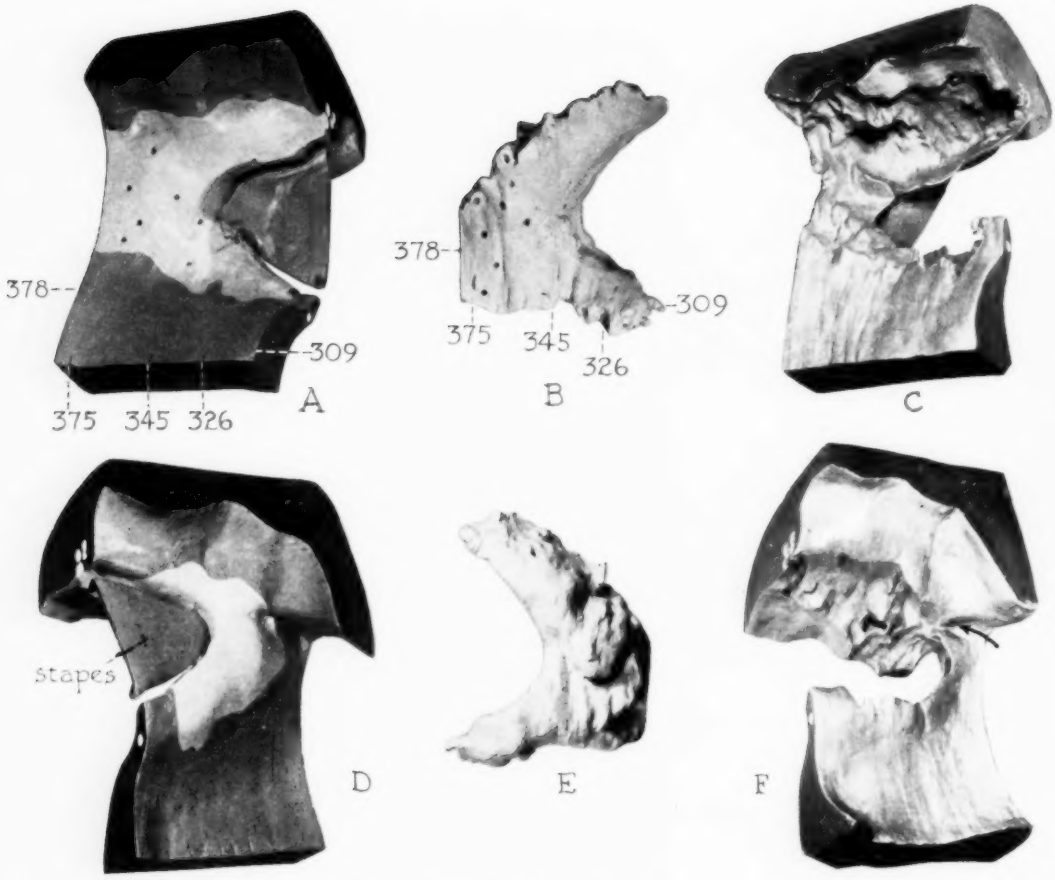

Plate I.—Photographs of three models of the fenestral area in an adult human ear, showing normal and otosclerotic bone, the latter surrounding approximately the ventral (anterior) half of the vestibular window; × 11. The numbered levels indicate the positions of the sections seen in plates II and III. The cut surfaces are shown in black. Figures *A*, *B* and *C* show the models turned to display the aspect representing the lateral or tympanic wall of the middle ear; figures *D*, *E* and *F* show the same models reversed to display the medial or labyrinthic wall of the labyrinth. Figures *A* and *D* show the pathologic bone (in light tone) and the normal bone (in darker tone) in which the former is embedded; figures *B* and *E* show the pathologic core modeled separately; figures *C* and *F*, the normal bone from which the core is enucleated. The anterior part of the stapes is modeled in figures *A* and *D*. The arrow in *F* points to the undestroyed vestibular extremity of the fissula ante fenestram.

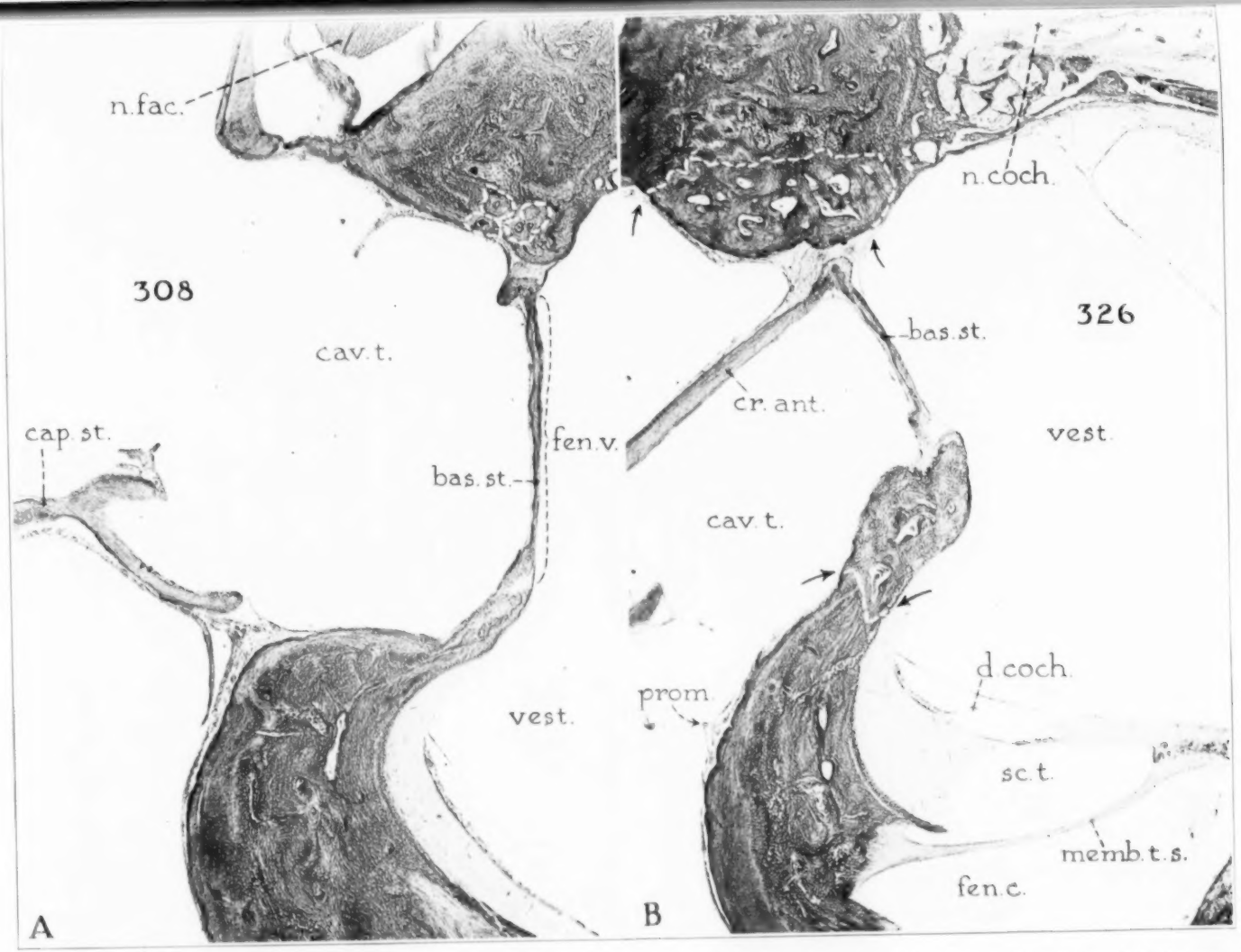

Plate II.—Photomicrographs of an area of sclerotic bone within the normal bone. The former, outlined in white, is marked by an arrow wherever it reaches the periosteal surface. The levels are indicated in plate I. *A* and *B*, sections (308 and 326) through the fenestra vestibuli. In *A* three small patches of pathologic bone are shown at the superior aspect of the window; in *B* such bone forms the entire wall of the window; × 20.

in relation to the whole bone, in a plane intermediate between vertical and horizontal. The models were prepared by the Born wax plate method from tracings made with an Edinger projection apparatus at a magnification of 125 diameters. The models consist of 73 plates representing sections between numbers 306 and 378; 44 of the wax plates represent separate sections, and the remaining 29 represent duplications of adjacent and closely similar sections.

OBSERVATIONS

With the exception of two relatively small foci situated between the main nodule and the cochlea, the new bone was limited to the recognized "site of predilection," in the lateral wall of the labyrinthine capsule, at the anterior margin of the oval window. As shown in the models, it consisted of an anterior chief portion from which lesser rootlike extensions reached partially around the superior and inferior margins of the window. The whole sclerotic area extended through 83 sections (from 306 to 388 inclusive), and its estimated length was 3.46 mm.; the models include the affected region through section 378. When seen as a separate enucleated mass, that surface which was directed toward the middle ear was found to be quite smooth (plate I *B*), and largely exposed so as to form part of the tympanic wall in front of the oval window (plate I *A*); but much of the deeper aspect, directed toward the internal ear or labyrinth, was irregularly lobulate (plate I *E*) and less exposed (plate I *D*); the lobulations produced considerable corresponding excavations in the normal bone (plate I *C* and *F*). The roots of the sclerotic mass formed the boundary of the oval window in its anterior half (section 326, plate II *B*;[2] model, plate I *A* and *D*), and yet its spread had not involved the stapedial ligament or the stapes. The roots terminated posteriorly in pointed tips (plate I *B* and *E*, and the corresponding hollows in *C* and *F*). The basal part of the mass (section 345, plate III *A*) was exposed to both the tympanic and the vestibular surfaces (plate I *A* and *D*), except at its anterior limit (section 375, plate III *B*) where it was exposed only on the tympanic side.

In sections, the diseased bone stained so deeply red with eosin that it was readily distinguishable from the normal bone surrounding it on

2. The abbreviations used in plates II, III and IV are as follows: *bas.st.*, basis stapedis (base or foot-plate of stapes); *cap.st.*, capitulum stapedis (head of stapes); *cav.t.*, cavum tympani (tympanic cavity or middle ear); *cr.ant.*, crus anterius (anterior crus); *duct.c.*, ductus cochlearis (cochlear duct); *fen.c.*, fenestra cochlearis (cochlear or round window); *fen.v.*, fenestra vestibuli (vestibular or oval window); *memb.t.s.*, membrana tympani secundaria (secondary tympanic membrane); *n.coch.*, nervus cochlearis (cochlear nerve); *n.fac.*, nervus facialis (facial nerve); *prom.*, promontorium (promontory); *sc.t.*, scala tympani; *sc.v.*, scala vestibuli; *vest.*, vestibulum (vestibule).

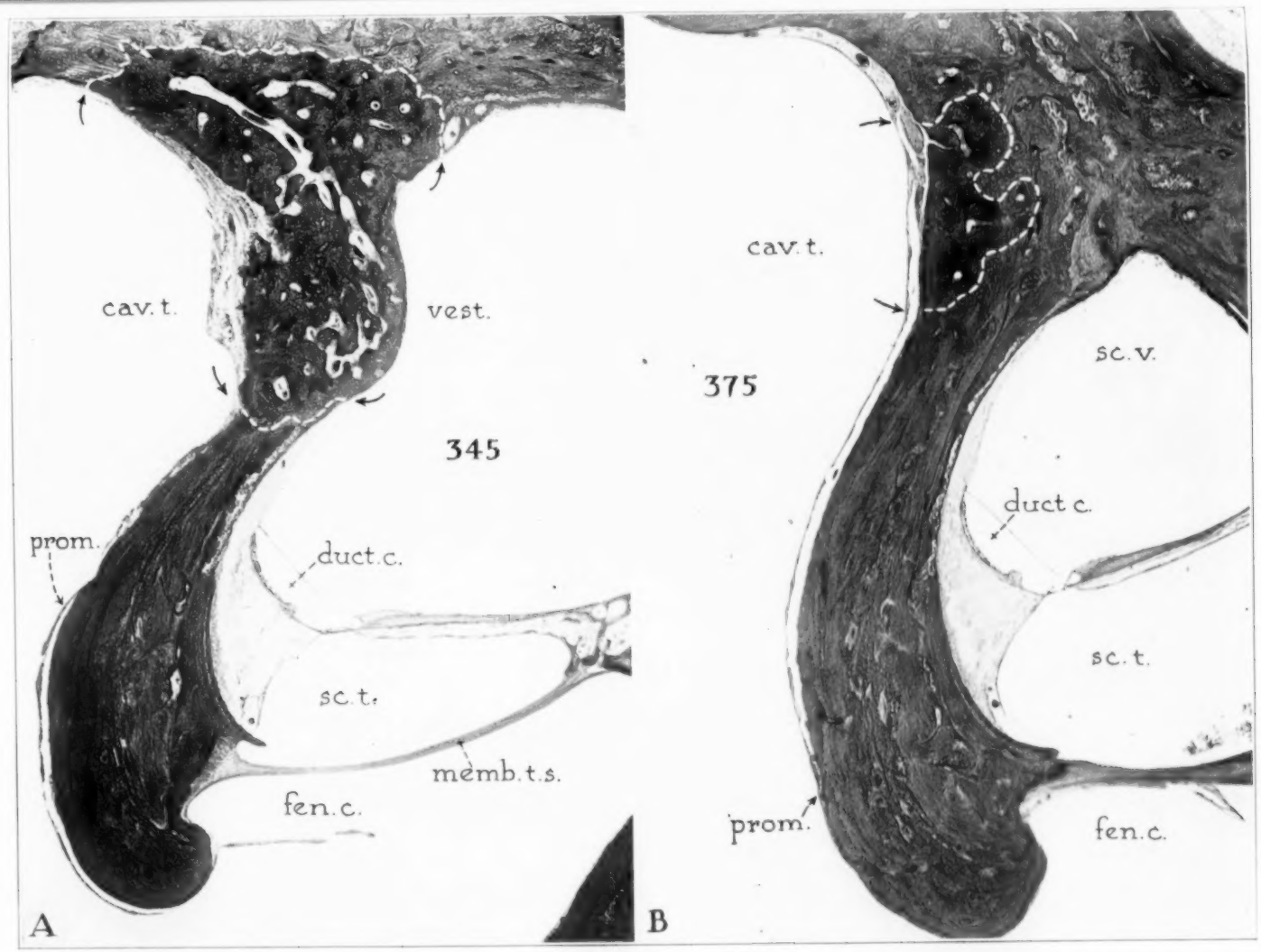

Plate III.—*A* and *B*, sections (345 and 375) through the fenestra cochleae, ventral to the level of the oval window. In *A* the vascular nature of the otosclerotic bone is evident; × 20.

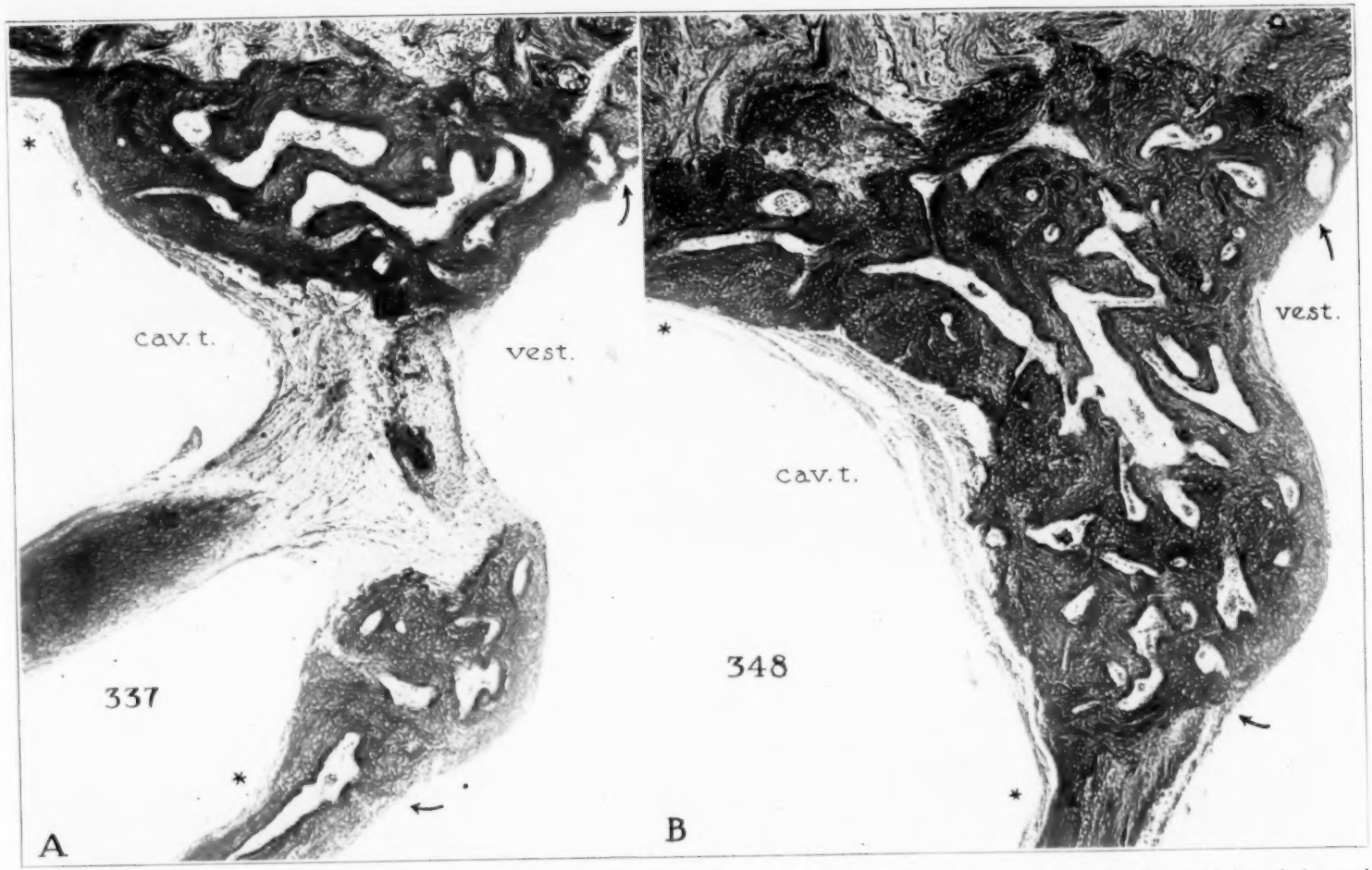

Plate IV.—*A*, section (337) through the spongious bone of the otosclerotic area, bordering on the superior and inferior margins of the oval window. *B*, section (348) through the porous sclerotic bone, where it forms the anterior boundary of the oval window (cf. plate III *A*) and borders on the vestibule and the tympanic cavity. In both sections the limits of the sclerotic bone on the vestibular aspect are distinct (note arrows) as on the tympanic aspect (near each asterisk) it is continuous with the perpetual lamellæ of the normal bone.

examination with the naked eye. This eosinophilic diseased bone was traversed, especially at its core (plates III *A* and IV *B*), by a network of broad anastomosing spaces each of which contained a large thin-walled blood vessel and a loosely arranged network of areolar tissue in which fat cells were rare. Space did not, in any section, predominate over bone, but it was sufficiently abundant to impart to the tissue the spongified appearance noted in the osteoporosis. The rarefied bone did not lack a definite pattern, but consisted of lamellar systems arranged around the vascular spaces. Generally, the line along which it bordered the normal bone was very distinct, and wavy or crenated in outline (plate IV *A* and *B*). The total absence of interglobular spaces (intrachondral bone) in the spongified nodule and the abundance of these in the adjacent bone further served to define the old and the new tissue.

The lamellae of the focus did not always remain concentrically grouped around a vessel, but instead, where the new adjoined the old bone, extended peripheralward to become continuous with lamellae of elongate periosteal type (plate IV *A* and *B* at each asterisk); in such instances the areas of deep and of light stain were not sharply defined.

The central cavities within the sclerotic bone were larger than the haversian canals in the adjacent bone, but much smaller than marrow spaces. The cavities (plate IV *A* and *B*) contained a fine meshwork of reticular tissue fibrils in which fat cells were extremely rare. The meshes of the reticulum contained very few blood cells; the reticulum within each space supported, usually, only one thin-walled vessel, which anastomosed with similar vessels as the containing cavities intercommunicated (see especially plates III *A* and IV *B*).

In the area of the rootlike masses, the blood vessels which entered and left the sclerotic bone were related principally to the haversian vessels; those which supplied the main mass, bordering on the vestibule and the tympanic cavity were, on the contrary, principally of periosteal origin. Specifically, of the twenty-six vessels which entered or left the roots, twenty were from the deep surface, four of them entering the pointed tips (section 308, plate II *A*); only six were of periosteal origin. Of the twenty-four which supplied the anterior undivided mass, eight were related to the surrounding bone and sixteen to the periosteum—all but two of the periosteal vessels originating from vessels on the tympanic, rather than the vestibular, side. Although a highly vascular tissue, its spread was not without restraint; it did not encroach appreciably on the space of the oval window or produce osteophytic projections; it did, however, obliterate all but the vestibular extremity of the fissula ante fenestram.

Neither osteoblasts nor osteoclasts were seen; there was no evidence of an inflammatory exudate.

SURGICAL IMPORTANCE OF THE MASTOID VEIN IN INFECTED LATERAL SINUS THROMBOSIS

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SAN FRANCISCO

In a survey of the literature on the surgical importance of the emissary vein, usually described as the external mastoid vein, one is impressed with the minimum amount of material presented. This is especially noticeable in the American publications. Somewhat more has been published in the foreign journals. There are few subjects in otolaryngology on which less has been written, particularly in regard to the surgical and clinical aspects. Whether this is due to a lack of interest or to the prevailing opinion that it is of no consequence, I am unable to state.

Cheatle [1] published the most comprehensive article (in English) on the mastoid vein that I have had access to. He discussed the anatomy of the mastoid vein, its variability and its vestigial character. His observations are based on a study of many hundreds of skulls. His study also included the mastoid foramen, with deductions therefrom as to its possible clinical and surgical importance. He specifically mentioned its surgical value and stated that "one can realize that if sepsis extends down the occipital or external jugular vein, or if the lateral sinus empties entirely through the mastoid vein, it would be necessary to ligate and excise the external jugular in the same way as the internal jugular is dealt with in infected lateral sinus thrombosis." He further stated that the vein may be infected from the lateral sinus, and that this would appear to be usual in those of greatest size (fig. 1).

Politzer mentioned edema and swelling that may occur over the vein in the presence of infection of the lateral sinus. This diagnostic observation has been published under the name of Griesinger's sign. In fracture of the posterior fossa importance has been placed on venous discoloration and swelling over the mastoid foramen and the course of the posterior auricular vessels. This is due to an injury to the vein or sinus. It has been described under the diagnostic term of Battle's sign.[2]

Read before the American Laryngological, Rhinological and Otological Society, in Chicago, June 8, 1933.

1. Cheatle, Arthur: J. Laryng. & Otol. **40**:633 (Oct.) 1925.

2. DaCosta, J. C.: Textbook of Modern Surgery, Philadelphia, W. B. Saunders Company, 1926, p. 748.

Woloschin [3] reported the minute anatomy of the emissary vein studied in 130 mastoids. His observations, with the statistical data presented, are based on a study of the vein with regard to its size and direction, as well as of the cellular characteristics of the mastoids. He

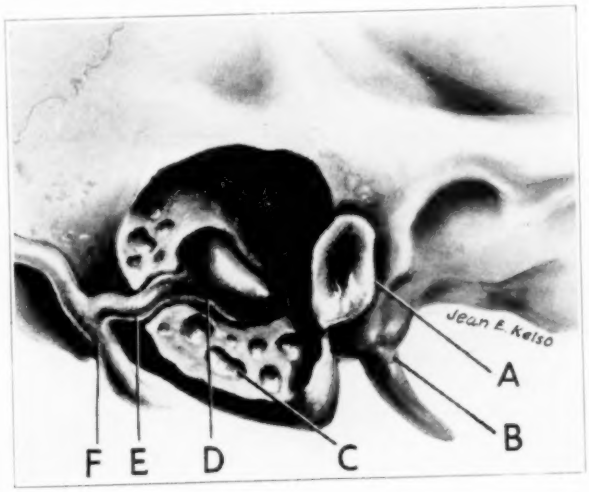


Fig. 1.—Drawing of a dissection done by me. It demonstrates: *A*, the external bony canal; *B*, the styloid process; *C*, pneumatic cells; *D*, the lateral sinus; *E*, a 6 mm. external mastoid vein, and *F*, the occipital vein.

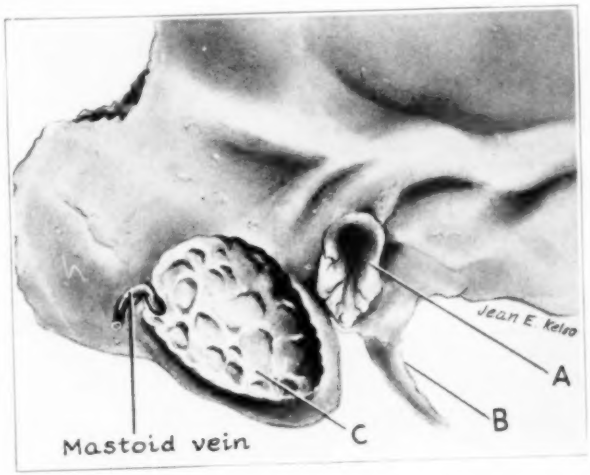


Fig. 2.—Drawing of a dissection done by me. It demonstrates a mastoid vein approximately 2 mm. in diameter. *A*, indicates the external bony canal; *B*, the styloid process, and *C*, the penumatic mastoid.

3. Woloschin, I. P.: Zur Topographie und Anatomie der V. Emissaria mastoidea beim Menschen in ihrer klinischen Bedeutung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:68, 1930.

also stated that, in his opinion, in the majority of cases the blood goes from the vein to the sinus, and only in a small percentage of cases from the sinus to the vein. I was unable to determine on what observations he made these deductions. Cunningham [4] stated that "the veins corresponding to the occipital artery are two, perhaps three in number. They drain the blood from the occipital portion of the scalp and open

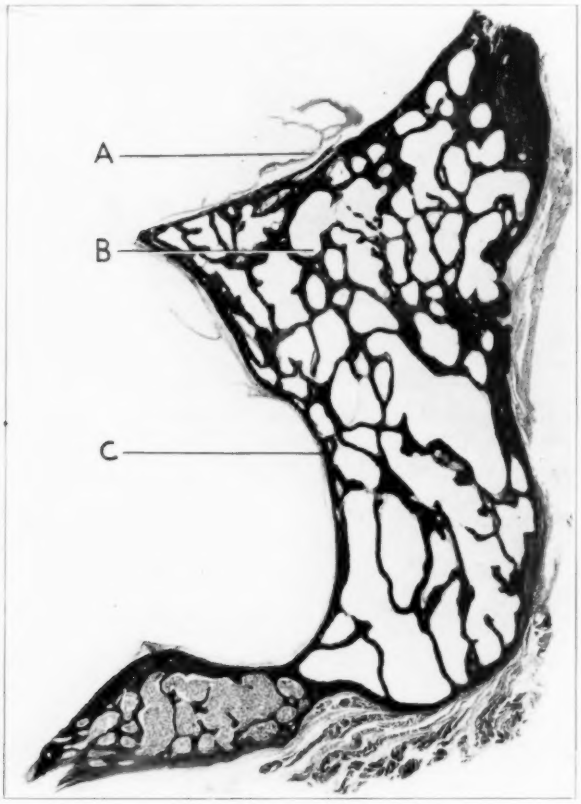


Fig. 3.—Photomicrograph of a section of a pneumatic mastoid. It shows: *A*, the intracranial fossa; *B*, pneumatic cells, and *C*, the lateral sinus wall. This section was cut at an angle so as to show as much of the mastoid and sinus as possible.

into the suboccipital plexus, which is drained by the vertebral and deep cervical veins. The most lateral of the occipital veins frequently communicates with the lateral sinus, through the mastoid foramen."

4. Cunningham: Manual of Practical Anatomy, New York, William Wood & Company, 1927, vol. 3, p. 66.

Friedman and Greenfield [5] described a case in which the mastoid vein was separately and directly the seat of a septic thrombosis resulting from infected mastoid cells in close contact with the vein (fig 2).

In a presentation of the importance of the emissary vein in infected lateral sinus and jugular thrombosis, a brief résumé of the pathology is necessary. A thrombus in the lateral sinus is the sequence of an infection in the mastoid cells extending either by continuity of tissue or by

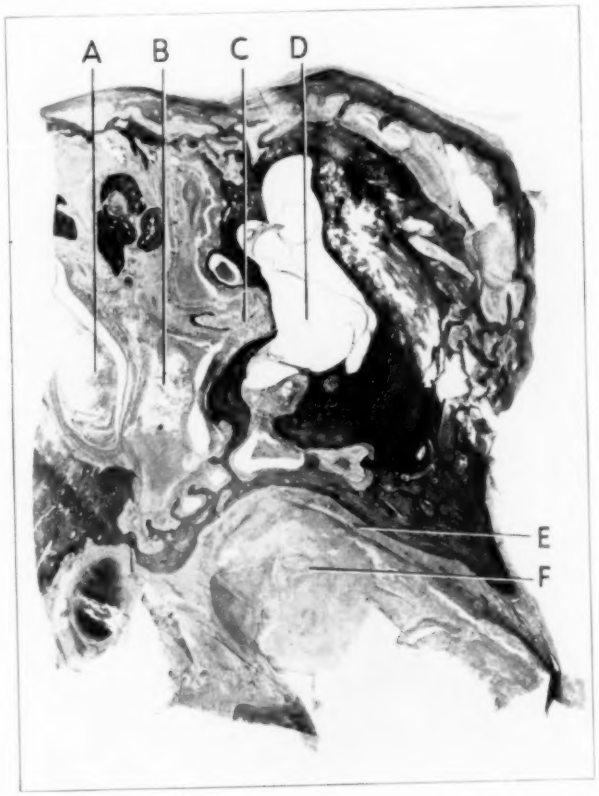


Fig. 4.—Photomicrograph of a pathologic section through the temporal bone. It demonstrates: *A*, the external auditory canal; *B*, a suppurative process in the middle ear; *C*, the round window; *D*, the labyrinth with saccule, utricle and a portion of the cochlea; *E*, the wall of the jugular bulb, and *F*, a thrombus in the jugular bulb.

venous pathway [6] (fig. 3). Primarily, a phlebitis develops. In time, owing to the involvement of the intima, with destruction of the endothelial lining, a thrombus forms (fig. 4). It is nature's method of

5. Friedman, J., and Greenfield, S.: Laryngoscope **33**:347 (May) 1923.

6. Kopetsky, S. J.: Otologic Surgery, New York, Paul B. Hoeber, Inc., 1929, p. 269.

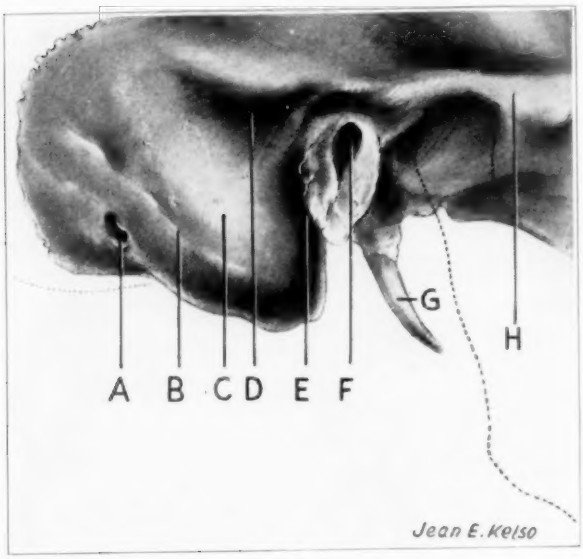


Fig. 5.—Drawing of a mastoid process showing a mastoid foramen about 2 mm. in diameter. *A* indicates the mastoid foramen; *B*, remains of the squamomastoid suture; *C*, the mastoid process; *D*, the mastoid fossa; *E*, the tympanomastoid fissure; *F*, the external acoustic meatus; *G*, the styloid process, and *H*, the zygomatic process.

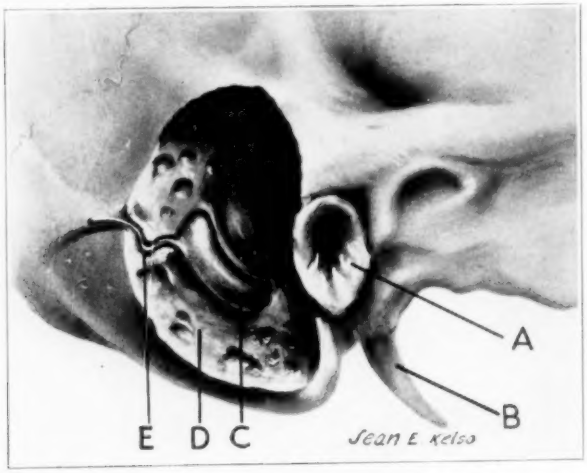


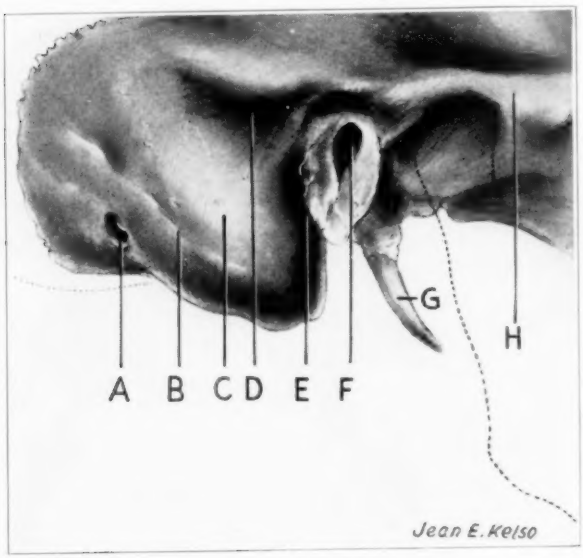
Fig. 6.—Drawing of a dissection done by me. It demonstrates the usual direction of the mastoid vein in relation to the lateral sinus. *A* indicates the external bony canal; *B*, the styloid process; *C*, the lateral sinus; *D*, the diplopneumatic tip, and *E*, the small mastoid vein directed posteriorly.

protecting the organism against a bacterial invasion of the general circulation. This is a pathologic principle applicable to vascular structures throughout the body, and should not be considered as being of any particular virtue possessed by the lateral sinus itself. Within the thrombus organisms are usually harbored. Their quantity and virulence and the resistance of the patient regulate the future destiny of the thrombus. The extent of the clot varies. It may remain localized, but as a rule it extends upward and downward for a variable distance. This

Fig. 7.—Drawing of a dissection done by me. It demonstrates the external mastoid vein emptying directly into the internal jugular. *A* indicates the posterior auricular vein; *B*, the external mastoid vein; *C*, the external jugular vein, and *D*, the internal jugular vein.

is quite natural, for nature in attempting to protect the human body against septic invasion always goes to extremes. It gives full measure of protection, provided that the potential resistance of the patient is such that the demands can be fulfilled. The thrombus may invade other sinuses such as the petrosals,[7] though this is not usual. In these sinuses

7. Brown, J. M., and Bowman, R. J.: Case Report of Phlebitis of the Lateral Sinus, Inferior Petrosal Sinus and Opposite Lateral Sinus with Post-Mortem Examination, Proc. West. Sect. Am. Laryng., Rhin. & Otol. Soc., 1933.



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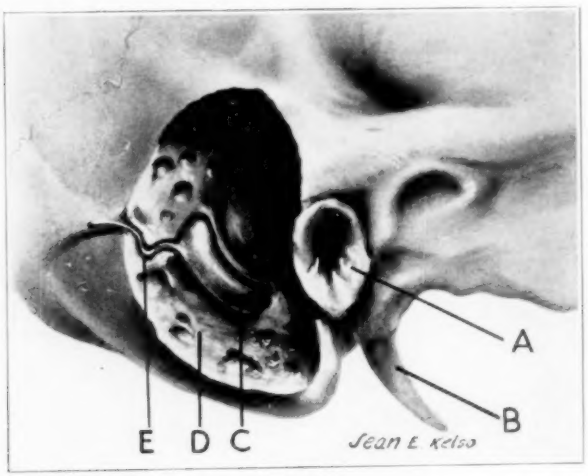

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the flow of blood is toward the lateral sinus. This probably lessens the chance of thrombus formation extending therein, unless the phlebitis is of such extent as to advance into these vascular channels. In my opinion, infected lateral sinus thrombosis does invade the external mastoid vein. The assumption that a thrombus extends into this structure may be accepted, considering the manner in which it varies in size, as has been determined from studies published in the literature as well as by personal anatomic observations. I can see no scientific reason why a

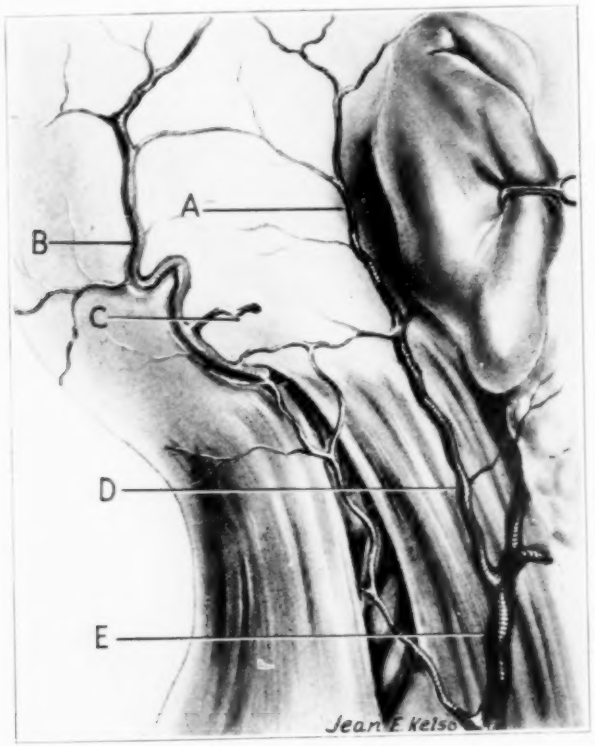

Fig. 8.—Drawing of a dissection done by me. It demonstrates the external mastoid vein emptying directly into the occipital vein. *A* indicates the posterior auricular vein; *B*, the occipital vein; *C*, the external mastoid vein, and *D*, the posterior auricular vein anastomosing with the branch of the facial vein to form the external jugular vein, *E*.

thrombus or a phlebitis, if sufficiently extensive, should not involve the emissary vein regardless of its size.

The stimulus for this work developed a number of years ago. While I was visiting a large postmortem clinic in one of the eastern hospitals, a case of infected lateral sinus thrombosis with internal jugular ligation came to autopsy. The external jugular vein appeared to be thrombosed

as well. Through the courtesy of the surgeon performing the autopsy, at my request a dissection was made of the external mastoid vein. It demonstrated that this vein was thrombosed and emptied directly into the external jugular vein, which in turn emptied into its normal portal of entry, the subclavian vein. It was my first and only observation of such a lesion. There developed in me a desire to know more about the anatomy of the parts involved. To my knowledge I have never had a

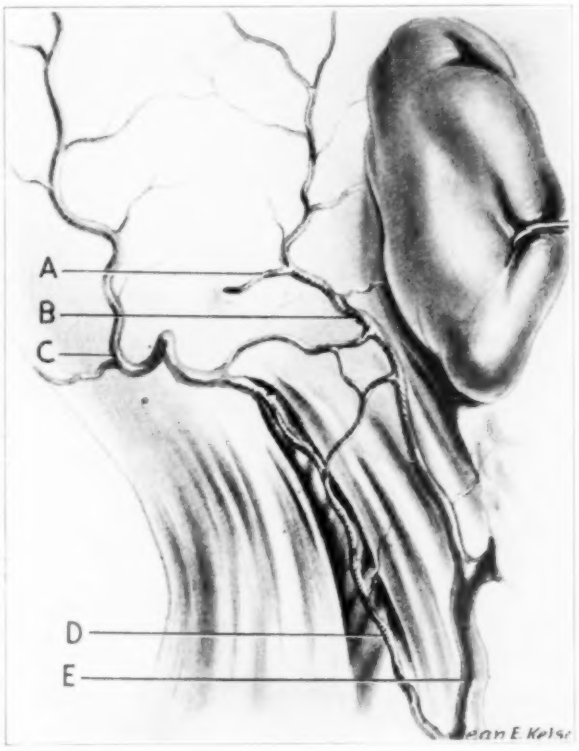

Fig. 9.—Drawing of a dissection done by me. It demonstrates the external mastoid vein emptying directly into the posterior auricular vein. *A* indicates the extrenal mastoid vein; *B*, the posterior auricular vein; *C*, the occipital vein, and *D*, the branch of the occipital vein communicating with the external jugular vein at *E*.

case of this type, but the anatomic studies presented here make me believe that the possibility is of practical clinical and surgical importance. The mortality from infected lateral sinus thrombosis is high. Might not some of these vascular characteristics account in a measure for the 30 per cent mortality?

In the observations made, a diameter of 2 mm. was taken as the arbitrary normal standard of the mastoid foramen (fig. 5). This was

accepted as a standard, since the diameter of the great majority of the veins studied fell below it. Also, it corresponds approximately with a definite mathematical unit of measurement, the line. In the dissections the direction of the vein (fig. 6), its point of entry into the venous circulation, its point of junction with the lateral sinus and the type of cells in the mastoid process were observed.

Table 1 is a report on 30 mastoid veins studied in cadavers.

Table 2 is a report on the type of cells encountered in the 30 mastoids studied.

TABLE 1.—*Postmortem Data on 30 Mastoid Veins*

Location of Veins	Size, Mm.	Anomalies	Absence of Vein	Direction	Relation to Other Veins	Point of Junction with Sinus
R. 19	4+	0	5	Upward..... 1 Horizontal... 2 Downward...11	Occipital..... 9 Posterior auricular.. 2 Internal jugular..... 1 External jugular.... 2	Internal.... 0 Posterior..12 External... 2
L. 11	2+	0	3	Upward..... 0 Horizontal... 2 Downward... 6	Occipital..... 5 Posterior auricular.. 2 Internal jugular..... 1 External jugular.... 0	Internal.... 0 Posterior.. 6 External... 2

TABLE 2.—*Types of Cells in the 30 Mastoids*

Location of Mastoids	Cellular Characteristics
R. 19	Sclerotic....4 Pneumatic....7 Diploic....5 Diplopneumatic....3
L. 11	Sclerotic....2 Pneumatic....5 Diploic....2 Diplopneumatic....2

If one is willing to assume that an infected thrombus may extend into the external mastoid vein, then from a clinical and surgical point of view one must be interested to observe how often this vein empties into the general systemic circulation other than by way of the internal jugular vein or one of its tributaries (fig. 7). Table 1 indicates that in 14 of the dissections the external mastoid was connected with a vascular structure that may or may not empty into the internal jugular vein. I refer to the occipital (fig. 8). In addition, 4 of the dissections demonstrated that the emissary vein emptied into the postauricular vein, either directly or through a main branch (fig. 9). By the union of the posterior auricular vein and a branch from the facial vein, the external jugular vein is formed (fig. 9). In 2 specimens it was demonstrated that the mastoid vein emptied directly into the external jugular vein (fig. 10). This vein, as is well known, empties into the subclavian vein. It was

noted that in those dissections relative to the occipital vein a definite venous plexus was usually encountered (fig. 11). This plexus was not studied as to its many ramifications. It may be safely stated that this plexus is prone to anastomose through its many vessels with any or all of the veins of the neck, either superficial or deep.

It is my opinion that attention should be given to the mastoid vein in all cases in which the presence of an infected lateral sinus is suspected.

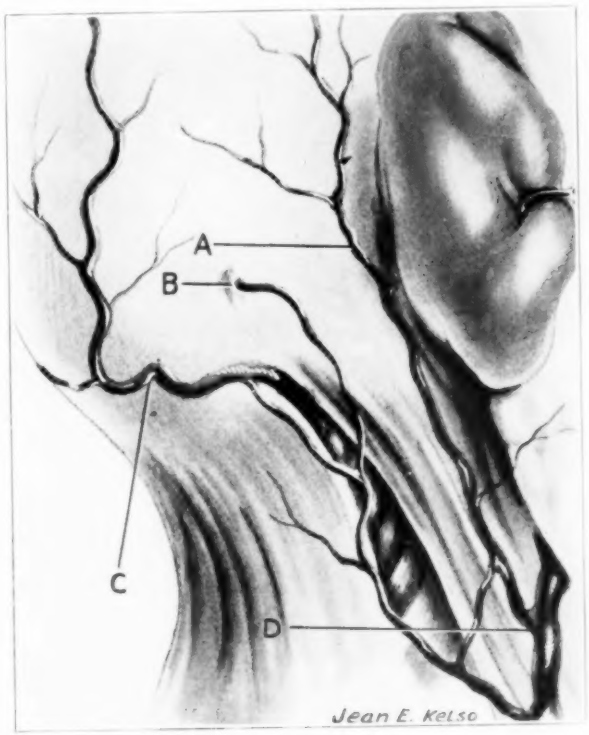

Fig. 10.—Drawing of a dissection done by me. It demonstrates the external mastoid vein emptying directly into the external jugular vein. *A* indicates the posterior auricular vein; *B*, the external mastoid vein; *C*, the occipital vein, and *D*, the external jugular vein.

From the publication of Kraus and Wirkner,[8] who studied the anatomy in conjunction with the roentgenologic findings, the conclusion is made that information can be obtained as to the size of the mastoid vein by roentgen studies of the foramen. In the surgical procedure might it not be well to expose the vein to determine the presence of a thrombus.

8. Kraus, L., and Wirkner, D. J.: Anatomische und roentgenologische Untersuchungen über das Emissarium mastoideum, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**: 270, 1927.

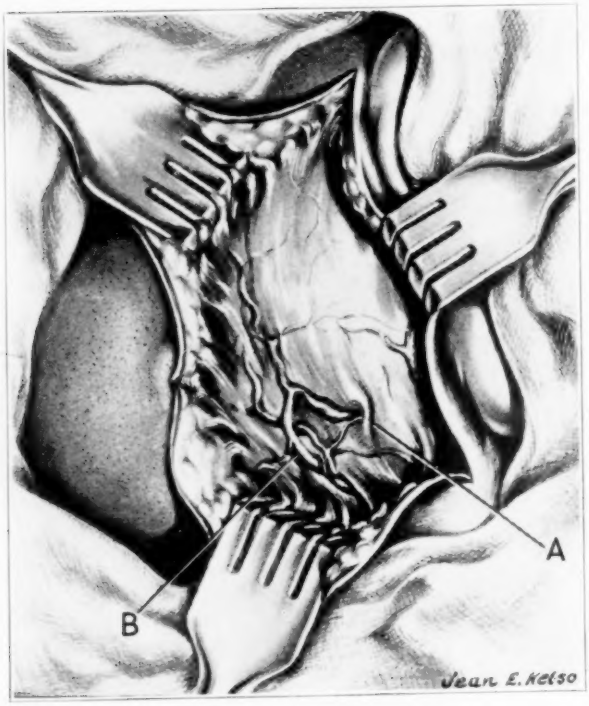

Fig. 11.—Drawing of a dissection done by me. It demonstrates the complexity of the occipital plexus as it lies beneath the trapesius or posterior border of the sternomastoid muscle. *A* indicates the external mastoid vein, and *B*, the occipital vein with plexus.

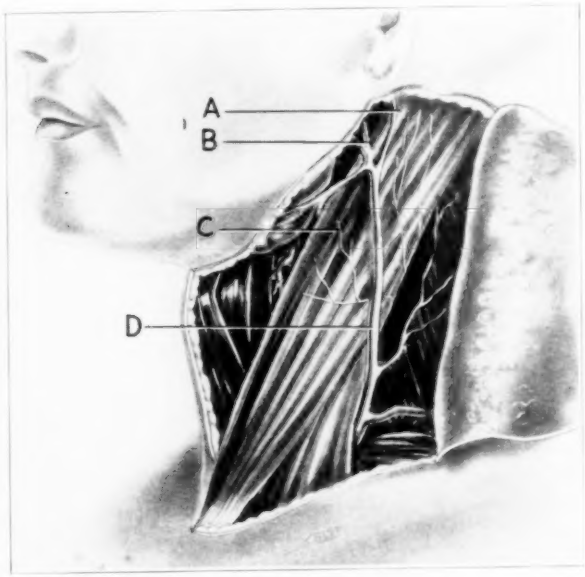

Fig. 12.—Drawing of a dissection done by me. It demonstrates the anatomic formation of the external jugular vein and its relation to the muscles of the neck. *A* indicates the posterior auricular vein; *B*, the posterior branches of the external facial vein; *C*, the sternomastoid muscle, and *D*, the external jugular vein.

and if a thrombus exists, to ligate the vein? By so doing a systemic invasion by a portal other than the internal jugular vein might be avoided.

CONCLUSIONS

1. The mastoid vein is frequently absent. In 25 per cent of the specimens studied such a condition occurred.

2. It has a variable point of entrance into the venous circulation.

3. It varies in size and direction.

4. It may be invaded in an infected lateral sinus thrombosis, either by continuity of tissue, that is by a phlebitis, or by extension of the thrombus directly.

5. It may account for some of the high mortality in infected sinus thrombosis.

6. It should be considered from a surgical point of view in all cases of infected lateral sinus thrombosis.

LARYNGEAL SCLEROMA IN A NATIVE MISSOURIAN

REPORT OF A CASE

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AND
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The first case of true scleroma of the larynx in a native of the United States was recorded by Watkins[1] in 1921. The patient was a Negro, aged 23, born in Baltimore. Watkins stated that the only other authentic cases reported in America up to that period were in immigrants. His patient had been a fireman on a ship which plied between Baltimore, the West Indies and Central America, where rhinoscleroma is said not to be uncommon.

The only instance of scleroma that we were able to find recorded as occurring in a native of the United States who had never been out of the country was reported by Figi[2] in 1928. The patient was a man, aged 26, born in New Jersey of Slavic parentage, who had a history of nasal discharge of eight years' and of hoarseness of two years' duration. He showed an extensive destructive lesion of the nose, pharynx and larynx. Radium treatment, potassium iodide, and an autogenous vaccine were used with striking improvement in the patient's condition.

REPORT OF CASE

The case which we wish to record occurred in a Mexican youth, aged 20, who was born and reared in Kansas City and who had never been away from that city.

Clinical History.—M. A., a single man, in June, 1931, began to have hoarseness and aphonia, with occasional spells of difficulty in deglutition. In September, 1932, a "cold" with cough developed, and for three or four days he was extremely hoarse and aphonic. The cough disappeared, but the hoarseness remained. On November 2, the patient had the first attack of asphyxia. When seen by one of us (N. J.) in the home, he was found gasping for air. He was cyanotic, and his entire body was covered with profuse perspiration. When the mouth and throat were examined with a tongue depressor, nothing could be found to cause such severe symptoms. His temperature was normal, and the mouth and pharynx

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1. Watkins, S. S.: Primary Scleroma of the Larynx in a Negro Born in Maryland, Surg., Gynec. & Obst. **33**:45 (July) 1921.

2. Figi, F. A.: Rhinoscleroma, Proc. Staff Meet., Mayo Clin. **3**:118, 1928.

appeared normal except for pyorrhea and dental caries. The nose showed hypertrophy of the turbinates. Morphine, ⅛ grain (0.008 Gm.) by hypodermic injection, gave him almost complete relief in about twenty minutes. On December 8, he was seen in a second attack almost identical with the first. At this time it was stated that almost any exertion on the part of the patient might cause an attack. On this occasion, the patient was found to be somewhat underweight, indolent and melancholic. The Wassermann test was negative. The larynx could not be visualized, and the patient refused to be removed to the hospital. A diagnosis of laryngeal syphilis was made, and he was given intravenous injections of sodium iodide which seemed to afford him some relief. On February 6, he had another attack similar to the earlier ones, and died with symptoms of suffocation before any help could be administered.

The history was essentially negative, except for measles, scarlet fever, mumps and whooping cough in childhood. Four years prior to death the patient had severe pyorrhea which never completely cleared up.

According to his family, he had discontinued school after completing the fourth grade; since then he had had no occupation. He was distinctly subnormal mentally and spent a great deal of time picking around in the garbage and rubbish at the city dumps. He had smoked from the age of 10 years and had used alcohol from the age of 15.

The family history was carefully investigated, and an accurate résumé of the cause of death and of the diseases of the entire family back to both of the great grandparents of the patient was given. None of his immediate family or remote ancestors had ever had a condition similar to that from which the patient died.

Necropsy.—The body was that of a moderately undernourished, somewhat anemic-appearing Mexican youth, aged 20. Both visceral and arterial embalming had been carried out, and the mouth and nose could not be thoroughly investigated, owing to the fact that extreme rigor mortis had set in and that the mouth was sewed shut by the embalmers. We were able to obtain permission for an investigation of only the cavities of the body, and it was therefore necessary to remove the organs of the neck and mouth from below. Nothing of noteworthy interest was found in the peritoneal cavity except marked visceral hyperemia. In the thoracic cavity, both lungs were found to be widely expanded and intensely hyperemic, and subserous hemorrhages were seen dotted here and there over the visceral and pleural surfaces. The trachea and bronchi contained frothy, blood-flecked fluid.

There was a very striking stenosis of the larynx, and the glottis when viewed from above was so small that it would scarcely admit a match. The lumen was decreased because of a dense, hard, seminodular growth which involved the submucosa of the larynx and extended from the region of the upper end of the cricoid up to the aryepiglottic fold, with considerable involvement of this fold. Anteriorly, the growth involved the entire laryngeal submucosa and extended from the base of the thyroid cartilage to the glosso-epiglottic fold. The mucosa of the epiglottis was also involved to some extent by this same type of submucosal thickening. This extreme submucous infiltration had caused an almost complete stenosis of the larynx. The mucosa itself was somewhat pitted in places and even showed slight surface erosion. Both of the vocal cords were thickened and distorted. The surface of the growth was slightly bosselated, and occasionally small granules could be seen projecting above the surfaces of the intact mucosa. The tracheal mucosa was not involved. On section, the growth was of pale grayish

white and markedly indurated. It was more or less homogeneous in appearance and appeared more like inflammatory fibrous tissue than neoplastic tissue. On cross-section, the cartilage of the epiglottis showed small areas replaced by this same granulomatous tissue, thus producing numerous breaks in the continuity of the cartilage proper.

The tongue and tonsillar ring showed nothing noteworthy grossly. Further investigation of the oral cavity was unsatisfactory. There was rather marked dental caries.

Histologic Pathology.—The squamous epithelium of the laryngeal mucosa showed thickening of the epidermis and superficial keratinization. Pus cells were found in the epidermis and on its surface. There was some downgrowth of the

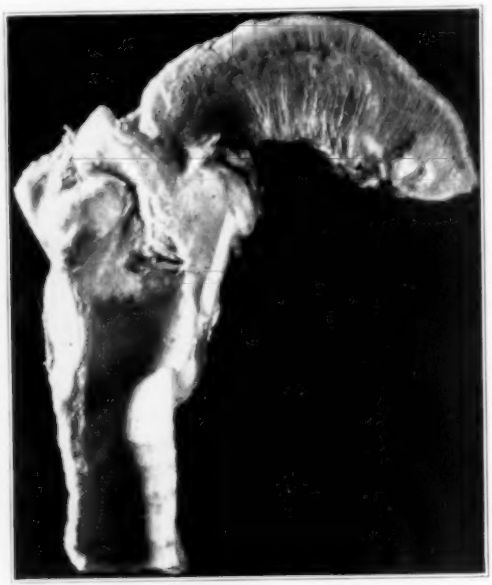

Fig. 1.—Longitudinal hemisection of the larynx, showing stenosis from scleroma.

rete pegs, which were frequently much more slender than normal and which often penetrated into a dense type of granulation tissue rich in inflammatory cells of many kinds. In this granulation tissue, large, pink-staining, oval and rounded hyaline bodies were seen, sometimes with an eccentrically placed nucleus and sometimes with a nucleus in the center, whereas in other instances no nucleus was present. In addition to the single homogeneous hyaline bodies, other hyaline masses were seen which gave the impression that they were made up of an agglutination of several smaller, rounded hyaline masses. Plasma cells, fibroblasts, round cells, polymorphonuclear neutrophils and young capillaries were present in abundance. Many of the capillaries were packed with pus cells. In addition to the findings in the granulation tissue, we found, sometimes in rather large numbers and sometimes only occasionally, large, foamy cells with vacuolated cytoplasm and irregular peripheral borders. These cells usually contained a small, pyknotic, centrally located nucleus and in all respects were identical with the typical

Mikulicz cells of scleroma. The granulation tissue extended deep into the underlying muscle and connective tissue, and heavy bundles of old, fragmented fibrous tissue could be seen separated by the granulomatous masses. Occasionally, glands and small groups of glands were encountered, some of them showing active secretion. With only the hematoxylin and eosin stain it was possible to discern accumulations of organisms in the foam cells and with Brown-Breen and Gram stains these foam cells were found to contain large numbers of organisms, for the most part gram-negative bacillary forms. In addition to these forms, other organisms were found in great numbers, and sometimes the cytoplasm of the foam cell seemed almost bursting with an overaccumulation of them. Moreover, scattered about in the interstitial tissue, singly and in small clusters, cocci and

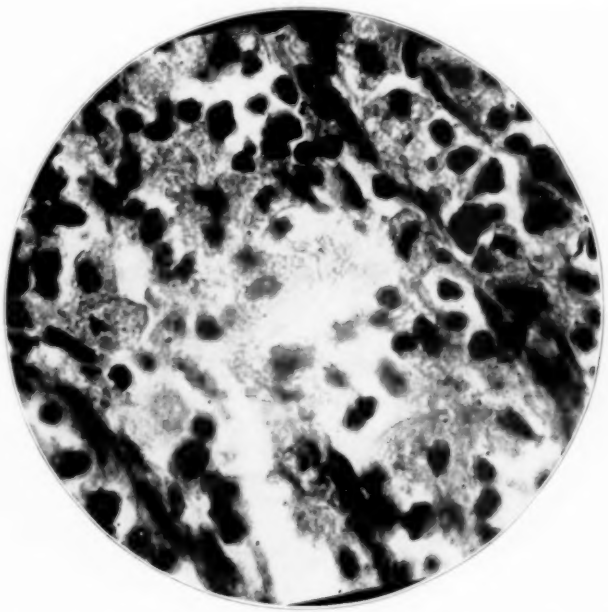

Fig. 2.—Oil immersion photomicrograph of the section from the submucosa of the larynx, showing large numbers of Mikulicz or foam cells.

bacillary forms were often seen. Those within the Mikulicz cells were gram-negative, and most of the extracellular organisms were likewise gram-negative.

Under the higher magnification, the tissue showed a striking breaking up and disorganization of connective tissue bundles by the massive granulomatous involvement. Occasionally, Mikulicz' cells were found entirely free from organisms. Levaditi stains showed no evidence of spirochetes. Sections were submitted to Dr. Robert Koritschoner of Kansas City and Dr. R. H. Jaffé of Chicago, both of whom concurred in the diagnosis of scleroma.

Aside from subpleural and subepicardial petechiae and marked hyperemia of the abdominal viscera, nothing of microscopic interest was seen. The lymph glands of the neck and mediastinum showed no evidence of scleroma, and the bronchi and trachea were also free from involvement.

COMMENT

Scleroma is common in Europe, particularly in the central and southeastern portion. In Poland, Austria and Rumania, it seems to be endemic. It has also been seen not infrequently in Russia, Eastern Asia, North Africa and Australia. It has been recorded as being present not infrequently in Central America, and Castellani [3] stated that in Guatemala it is not at all uncommon. Dr. Esteban Reyes [4] has personally seen about three hundred cases in San Salvador, which lies between Guatemala and Spanish Honduras, and he made the interesting observation that scleroma frequently developed in workers in "deep blue analine dyes," and that he believed that the dye contained the causative organism. In North America, as has been stated, the disease is extremely rare. Only one earlier case has been recorded in a native North American who had never been out of the country.

Most writers closely associate scleroma with unhygienic conditions, and accoring to Figi and Thompson [5] it is a disease of the "great unwashed." This theory is particularly interesting in regard to our case, since our patient was the one member of his family who had slovenly, filthy habits, and who was frequently found picking his meals from garbage about the city dump. Incidentally, a careful and painstaking family history gave no record of a similar condition in other members of his family. The disease is considered by most writers to be infectious; one of the most striking instances was that recorded by Lasagna [6] of Italy, who recorded fifteen cases in one town, the majority of them occurring in members of a family living in one domicile.

There has been considerable variance of opinion as to whether the organism described by von Frisch and named Bacillus rhinoscleromatis is the causative factor in scleroma. Nevertheless, the most recent work done by Figi and Thompson tends to confirm the opinion that the Frisch bacillus is the true etiologic factor. According to these investigators, the Frisch bacillus can be isolated in all cases. Moreover, lesions simulating those seen in rhinoscleroma have been produced in experimental animals by injection of the bacilli, and the organism has again been recovered from these lesions.

Scleroma is often mistaken for syphilis, and in many of the American cases reported as involving the larynx, as in our case and in Watkins' case, the condition was diagnosed as tertiary syphilis until necropsy

3. Castellani, quoted by Figi and Thompson.[5]

4. Reyes, Estaban, in discussion of paper by Alderson, H. E.: Scleroma, Arch. Dermat. & Syph. **26**:643 (Oct.) 1932.

5. Figi, F. A., and Thompson, L.: Rhinoscleroma, J. A. M. A. **91**:637 (Sept. 1) 1928.

6. Lasagna, F.: Infective Scleroma of the Respiratory Passages, J. Laryng. & Otol. **40**:369, 1925.

revealed the true identity of the lesion. Watkins stated that scleroma may at times be confused with tuberculosis, leprosy and malignant disease, and he held that marked chronicity, lack of destruction of the tissues and of pain, ivory-like hardness and a European nationality of the patient favor the diagnosis of scleroma, but that only microscopic study of the tissue makes this diagnosis certain.

The disease may start in the nose, pharynx or larynx, appearing as dense, remarkably indurated, cartilage-like, bosselated swellings. The process may extend to the lip, palate, pharynx, larynx and trachea, and has even been observed around the bronchi (Jaffé [7]).

The histologic pathology is relatively characteristic and depends on the presence of a granulomatous type of productive inflammatory reaction in which two distinct microscopic entities are present; i. e., the large, vacuolated foam cells of Mikulicz which often contain large numbers of Frisch bacilli and the red hyaline bodies of Unna, sometimes called Russell-fuchsin bodies. Considerable work has recently been done on the complement-fixation test, which, according to Elbert, Feldmann and Gerkes,[8] is of great value in diagnosis, especially in early and latent cases.

CONCLUSION

A case of scleroma of the larynx in a Mexican youth, aged 20, who had never been outside of Kansas City, Mo., is recorded. We were able to find a record of only one other case occurring in a native of the United States who had never been outside the country. The patient was mentally subnormal and had extremely filthy habits. The diagnosis during life was tertiary syphilis, and the true nature of the condition was not suspected until the microscopic sections were studied. In the histologic preparations, typical Mikulicz cells containing bacilli morphologically similar to B. rhinoscleromatis, and hyaline bodies of Unna were present. Bacteriologic studies and complement-fixation tests were impossible because of the embalming.

7. Jaffé, R. H., in discussion of Crowe, S. M.: Rhinoscleroma of the Larynx and Pharynx in Two Immigrants, Arch. Path. **5**:1125 (June) 1928.

8. Elbert, Feldmann and Gerkes: Centralbl. f. Bakt. (Abt. 1) **101**:384, 1927.

PLASMA CELL GRANULOMA SECONDARY TO GENERALIZED SEPTICEMIA IN A CASE OF NASAL CARCINOMA

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NOAH D. FABRICANT, M.D.

AND

GEORGE MILLES, M.D.

CHICAGO

From a consideration of the literature it appears that there is much confusion as to the significance of a cellular response predominantly of plasma cells such as one occasionally encounters in generalized septicemia. The difficulty is in separating or identifying the response of the lymph glands observed in cases of this type, as well as the granulomatous lesions encountered in the other tissues, from malignant conditions on the one hand and malignant lymphomas, particularly Hodgkin's disease, on the other.

The response of the lymph nodes of the body to any stimulus depends on the degree of severity of the stimulus, on the length of time the stimulus continues to act and on the susceptibility to stimulation of the various elements composing the lymph nodes, i. e., the reaction threshold of the components. The degree of severity of the stimulus is dependent on the concentration in which the stimulating agent reaches the lymph nodes and on the nature of that agent. This, together with the length of the time through which it acts, constitutes the exogenous factor. The susceptibility of the various elements of the lymph nodes is notably variable, a variability which is probably qualitative as well as quantitative.

The lymphoblastic tissue responds to a mild stimulus acting over a long period by the proliferation of mature lymphocytes. If the stimulus is increased, the response may be dominated by immature, or irritation, forms. A response which is colored by the nature of the stimulus to a certain extent and is seen commonly with rather severe stimuli acting over a long period is the plasma cell proliferation.

The response of the reticulum cells may be of several types. The first, resulting in general from mild or moderate stimuli acting over a

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variable period of time, consists in a simple swelling and proliferation of these cells. Secondly, a more violent stimulus may give rise to the formation of large, pleomorphic, free or attached forms and occasionally to mononucleated or multinucleated giant cells indistinguishable in some instances from the type seen in Hodgkin's disease. Add to these features the not infrequent eosinophilic infiltration encountered in chronic lymphadenitis, and a picture may result which closely resembles Hodgkin's disease.

The recent literature on this general subject is rather scanty, and only a few papers can be found with special reference to the morphologic relations to Hodgkin's disease. Apparently the formation of granulomas in the upper respiratory tract and elsewhere composed of plasmocellular elements is an extremely rare manifestation. The interpretation of this type of cellular response is so varied as to result in reports appearing in the literature, especially the European, under such a large number of titles as to make exhaustive analysis of the literature impossible. Earlier descriptions of this condition were presented by Schridde,[1] Pirone,[2] Alagna,[3] von Werdt,[4] Hedinger[5] and Vogt.[6] Frank and Kusunoki[7] described a plasmocellular granuloma of the neck with tumors of the nasal mucosa, which presented the picture of a "lymphoma." Later, Wachter,[8] Hückel,[9] Facchini and Scala[10] and Oppikofer[11] reported cases of "plasmocytomas" of the nasal passage. In the description by Rössle,[12] who reported a malignant

1. Schridde, H.: Weitere Untersuchungen über die Körnelungen der Plasmazellen, Centralbl. f. allg. Path. u. path. Anat. **16**:432, 1905.

2. Pirone: Sur les celluies plasmatiques, Folia haemat. **7**:339, 1909.

3. Alagna, G.: Die Plasmazellen bei Ohren-, Nasen- und Kehlkopf-Krankheiten, Virchows Arch. f. path. Anat. **204**:136, 1911.

4. von Werdt: Zur Kenntnis des Plasmocytoms, Frankfurt. Ztschr. f. Path. **6**:180, 1911.

5. Hedinger: Zur Frage des Plasmocytoms, Frankfurt. Ztschr. f. Path. **7**:343, 1911.

6. Vogt: Granulomatosis plasmocellularis colli, Frankfurt. Ztschr. f. Path. **10**:129, 1912.

7. Frank and Kusunoki: Ueber ein plasmozelluläres Granulom unter dem Bilde von Lymphomen der Halslymphdrüsen mit geschwulstartigen Knoten in der Nasenschleimhaut, Virchows Arch. f. path. Anat. **212**:391, 1913.

8. Wachter: Ein Fall von multiplem Plasmocytom der oberen Luftwege, Arch. f. Laryng. **28**:69, 1914.

9. Hückel: Ueber ein Plasmocytom des Nasenrachenraumes, Virchows Arch. f. path. Anat. **264**:172, 1927.

10. Facchini and Scala: Ueber einen Fall von Fibromyxoplasmocytom der Nasenhöhle, Arch. ital. di otol. **36**:331, 1925.

11. Oppikofer: Das Plasmocytom, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **23**:574, 1926.

12. Rössle, R.: Malignes Plasmocytom des Nasenrachenraumes, Schweiz. med. Wchnschr. **56**:302, 1926.

plasmocellular tumor of the nose, and that by Hajek,[13] who was astounded when he found two different types of tumors occurring in the sinuses of the same person, we find portrayed cases typifying the disease.

That these tumors are not common is shown by the presence of five cases in a period of twenty-five years (1905-1930), as evidenced in the work of Denker and Kahler.[14] Wallbach[15] viewed the general response of the lymph glands as differing even from one region to another. Osborne[16] cited a case in which cervical lymphadenitis, which on three occasions was diagnosed as Hodgkin's disease from biopsy specimens, apparently resulted from an apical infection, and cleared up completely following extraction of the tooth and resection of one gland.

In the case reported here, a rapidly growing anaplastic type of carcinoma apparently arising in the nares gave rise to retropharyngeal and cervical metastases. Secondary infection of the metastases to the lymph gland resulted in a virulent septicemia. The resultant generalized response of the lymph nodes illustrates an unusual response.

REPORT OF CASE

History.—A white man, aged 23, was admitted to the Research and Educational Hospital on Aug. 13, 1932. His chief complaints were severe pain limited to the right side and back of the head, a persistent pruritus extending over the right side of his nose and lips, a tremendous swelling of the neck, most marked on the right side, an obstruction to nasal breathing and a slurred, garbled speech.

The patient was well until April, 1931, when severe pain involving the right side of the head developed. Shortly thereafter progressive enlargement of the cervical glands was noted. In July, 1931, he had a nasal operation, and in December of the same year the pronounced cervical adenopathy was treated with radium. The cervical swelling disappeared under this treatment and remained in abeyance for one month, when it recurred. Roentgen therapy was instituted but without affording the patient relief from pain and without effect on the cervical adenopathy.

Examination.—On admission to the Research and Educational Hospital, the patient's marked emaciation and semicyanotic skin were noted as well as a marked hard diffuse cervical adenopathy (fig. 1). The right side of the nose was found completely obstructed by firm masses, and the left side was partially obstructed by soft tumors of a polypoid nature. About two thirds of the soft and hard palate on the right side was swollen and prominent, giving the impression of a nasopharyngeal mass bulging forward and downward. The mucosa was thick

13. Hajek: Zwei verschiedene, bisher nicht beschriebene Tumorarten der Stirnhöhle und des Siebbeinlabyrinthes in ein und demselben Individuum, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **23**:465, 1926.

14. Denker, A., and Kahler, O., quoted by Bröste, K.: Le plasmocytome des voies aériennes supérieures, Acta otolaryng. **18**:31, 1932.

15. Wallbach, G.: Zur Frage über die korrelativen Reaktionen von Lymphknoten und lockerem Bindegewebe, Folia haemat. **47**:329, 1932.

16. Osborne, O. T.: Disease of the Lymphatic Glands Caused by Tooth Infection, M. J. & Rec. **124**:598 (Nov. 17) 1926.

and dry; dental caries was marked, and oral hygiene much neglected. Multiple large lymph nodes of varying size were present in the axillae and groins. The liver was palpable 3 fingerbreadths below the costal margin, but the spleen was not palpable. In other respects the physical examination gave negative results. A tentative diagnosis of lymphosarcoma or Hodgkin's disease was made.

The blood picture disclosed 3,850,000 red cells and 47,500 leukocytes. The differential count showed 1.10 per cent lymphocytes, 2.05 per cent large mononuclear and transitional cells, 82 per cent polymorphonuclear neutrophils and

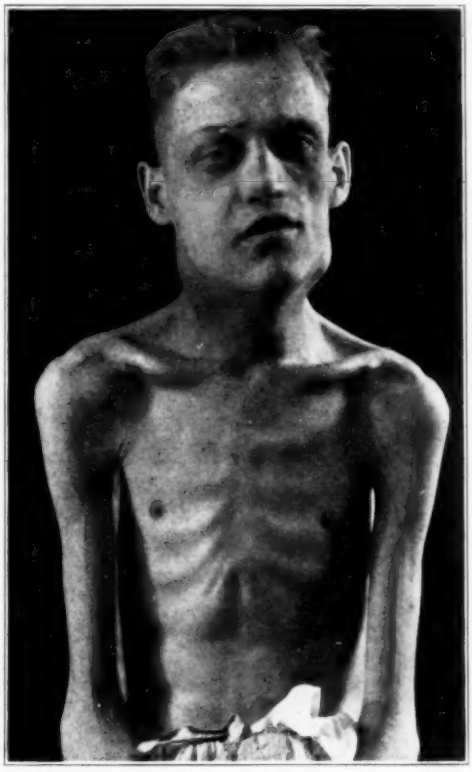

Fig. 1.—Appearance of the patient on his admission to the hospital, showing marked emaciation and cervical adenopathy.

14 per cent eosinophils. In over a thousand cells 1 myeloblast was seen. The urine contained blood, 1 plus; albumin, 4 plus, and numerous granular casts. Roentgenograms disclosed an increased density over the right maxilla and nasal cavity, with involvement of the bone.

Biopsy of a specimen taken from the right nares disclosed an edematous polyp with much secondary infection. An inguinal gland removed (fig. 2) at this time disclosed the picture of chronic lymphadenitis with numerous eosinophils scattered throughout the section. A lymph node removed from the cervical region revealed moderate fibrous proliferation with hyalinization, dense infiltration with plasma cells and eosinophils and marked proliferation of reticulum cells. Cells of giant

proportions with one or two nuclei were scattered through the section. A diagnosis of Hodgkin's disease was made as a result of the study of this section.

The clinical course in the hospital was progressively downward, and was characterized by severe pain. The temperature ranged from 98.6 to 102 F. On September 21, about one month after the patient's admission to the hospital, an obstructive dyspnea developed, which increased in severity until October 11, when a tracheotomy was performed. The patient died one week later.

From the clinical standpoint, the diagnosis was obscure, and the biopsy only added to the confusion. Hodgkin's disease was clinically considered most probable in spite of the fact that pain, a very unusual symptom in cervical Hodgkin's disease, was a predominant complaint. The sections taken for biopsy were considered to be examples of atypical Hodgkin's disease because of the marked

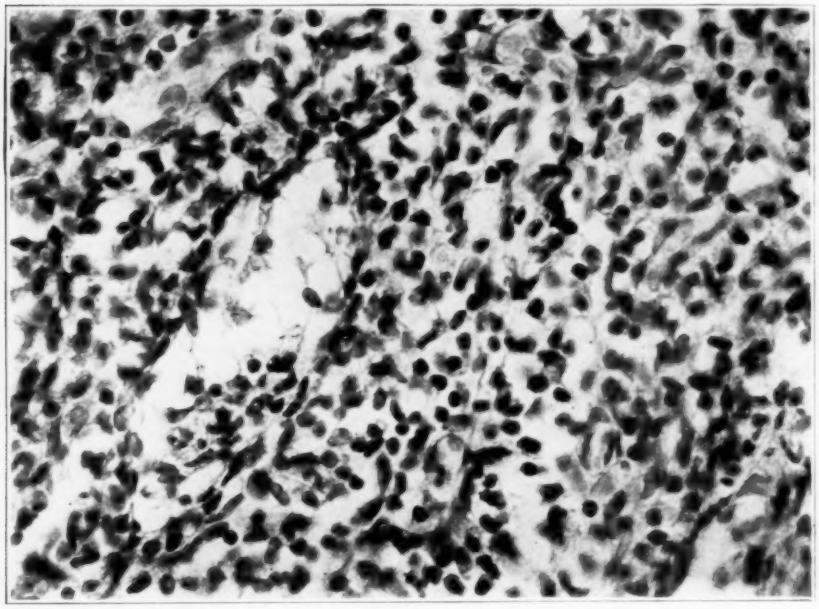

Fig. 2.—Inguinal lymph node, showing marked swelling and proliferation of the reticulum cells and numerous plasma cells and eosinophils; reduced from a magnification of × 800.

response of the reticulum cells in the presence of cells resembling Sternberg-Reed giant cells and marked eosinophilic infiltration and in spite of the large numbers of plasma cells.

Autopsy.—At autopsy the body was characterized by emaciation and a gray-white pallor of the skin. The soft palate and the posterior pharyngeal wall bulged markedly, and the upper half of the neck on both sides was the seat of a lobulated, irregular, firm swelling. A small amount of rather thick, creamy pus could be expressed from the incision where the specimen was taken for the last biopsy, and in the midline a tracheotomy opening was present. Shotlike, firm nodules were palpable in the supraclavicular, axillary and inguinal regions on both sides. The right antrum contained a few centimeters of thin, milky fluid and a single lobular

polypoid tumor mass 1 cm. in diameter. The left antrum contained a few cubic centimeters of thin, straw-colored, serous fluid. The soft palate averaged 1 cm. in thickness, and on section varied in color from gray to grayish tan and was of the consistency of soft rubber. Incision into the bulging pharyngeal wall released a considerable amount of thick, grayish-green, purulent material and exposed a poorly defined cavity lined by grayish-tan, friable tissue. From the retropharyngeal cavity thus opened a tract could be traced into the mass in the left side of the neck, from which pus could be expressed. Posteriorly, the bony wall, made up of the first and second cervical vertebrae, was necrotic and broken down, and a pathway into the spinal canal was easily made with a probe.

The tumor mass in the neck was made up of lobulated, firmly matted lymph nodes the centers of which were necrotic, and some were filled with thick, creamy

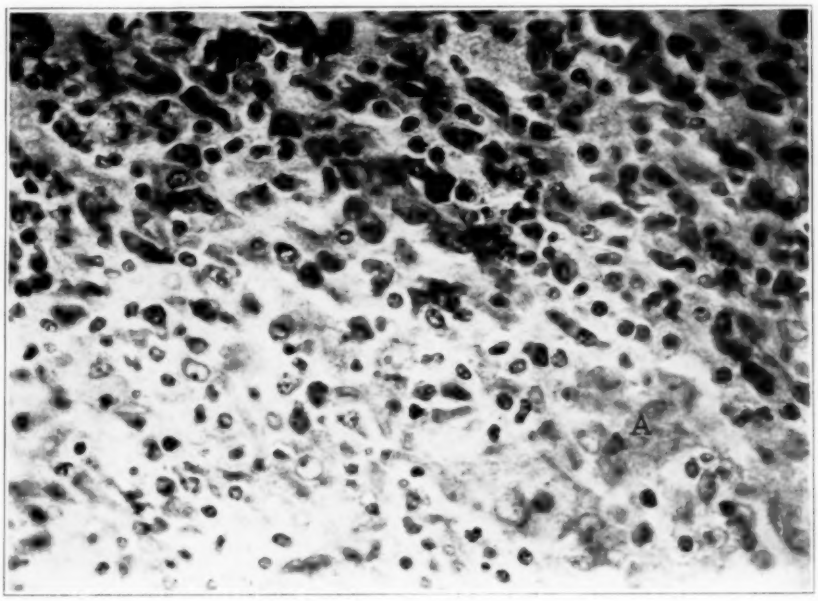

Fig. 3.—Nasal tissue, showing zones of tumor cells (*A*), surrounded by a dense infiltration with plasma cells, eosinophils and bizarre mononuclear and binuclear forms of macrophages; reduced from a magnification of × 800.

pus and others with a thin, serous exudate. Lymph nodes along the trachea and about the hilus of the lungs were enlarged to 3 by 1 by 0.5 cm., and were firm, and varied in color from grayish tan to dark gray. The abdominal lymph glands, including the periportal and peripancreatic group, were enlarged to 2 cm. in size and were firm and grayish tan. A confluent bronchopneumonia was apparent.

Bacteriologic Studies.—Blood, freshly drawn during life, was allowed to clot, and the blood clot was broken up and placed in veal infusion dextrose broth. The material was incubated for five days, at which time there appeared to be finely granular elements clinging to the wall of the flask. Smears made showed short, slender, gram-positive rods with occasional coccoid forms. Blood agar plates incubated with a drop of culture from the flask showed the same slender forms,

most of which were very short, resembling disintegrated rods and short coccoid bodies.

Autopsy Material.—Direct smears from both the free pus and the lymph node showed many typical chains of streptococci with good phagocytosis. No other organisms were seen. Blood agar plates inoculated from both yielded a pure culture of hemolytic streptococci in which two types of colony were present: (1) a small, smooth, glossy colony with a wide, clear zone of hemolysis and (2) a flat, dull, gray, rough colony with a wide zone of hemolysis. Colonies transferred to dextrose broth, incubated from eighteen to twenty-four hours and placed on blood agar showed both rough and smooth forms. Smears from the broth and blood agar cultures showed a variety of peculiar forms from single large cocci

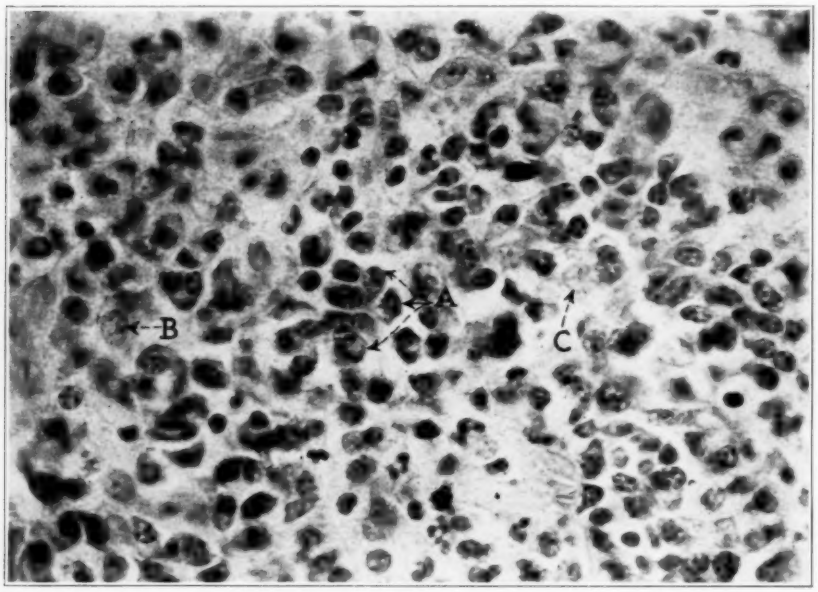

Fig. 4.—Cervical lymph node, showing: *A*, plasma cells; *B*, large mononuclear cells, and *C*, binuclear cells; reduced from a magnification of × 850.

to rods and typical chains showing bacillary elements or distorted large terminal cocci. When the organisms were transferred daily, an interesting picture was seen. Usually a typical streptococcic growth, granular in clear broth, was obtained, but frequently it resembled a staphylococcic culture with uniform dense nongranular clouding. Smears made from these tubes showed many bizarre forms, including grapelike clusters. Transfer of inoculums from these cultures to blood agar plates gave a pure growth of streptococci. The organism was pathogenic for rats and mice but not for rabbits. Thus a dissociable, pleomorphic, hemolytic streptococcus was demonstrated in the antemortem blood as well as in the pus and lymph nodes obtained at autopsy.

Microscopic Picture.—Tissue from the right nares (fig. 3) disclosed some superficial ulceration and dense infiltration of the subepithelial stroma by plasma cells, eosinophils, macrophages and neutrophilic polymorphonuclear leukocytes. Irregu-

larly distributed through the tissue were rather poorly defined sheets of anaplastic epithelial cells having a moderate amount of pale granular cytoplasm and single or occasionally multiple nuclei with finely granular chromatin and a prominent, deeply stained, oxyphilic nucleolus.

Section through the soft palate showed much the same picture with an additional feature of numerous focal areas of necrosis. In some sections of the cervical lymph nodes the normal structure was partially or completely replaced by similar tissue.

In other sections of the cervical lymph nodes (fig. 4) and of the abdominal lymph glands (fig. 5) the normal architecture was obscured by a diffuse proliferation of lymphoid cells and plasma cells, with many large mononuclear cells and a scattering of eosinophils. The dilated sinuses contained numerous mononuclear

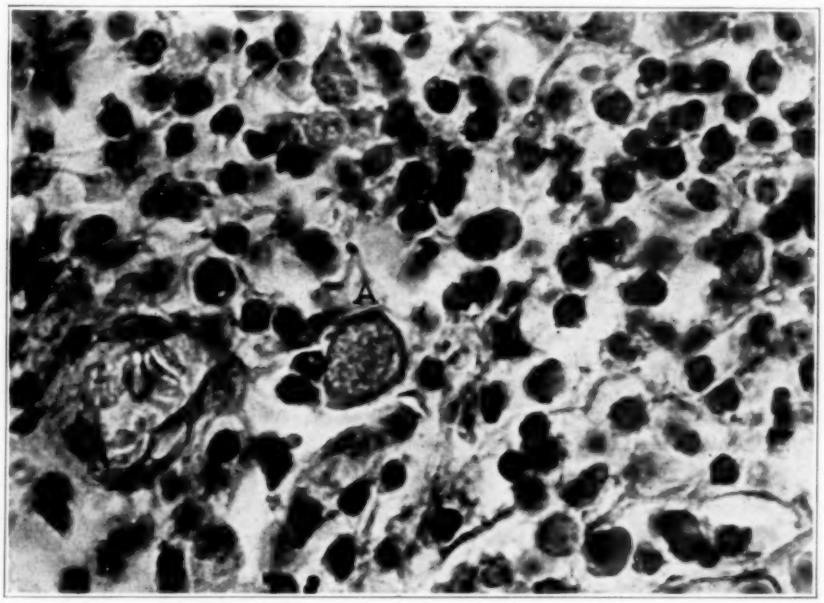

Fig. 5.—Abdominal lymph nodes, showing numerous plasma cells, eosinophils and bizarre forms such as *A;* reduced from a magnification of × 1,450.

cells and an occasional large cell with one, two or three nuclei. The reticulum cells were swollen and prominent. A similar picture was noted in the mediastinal glands.

In the spleen the sinuses were dilated and filled with eosinophils, plasma cells and macrophages. Occasional plasma cells were found in the sinuses of the liver, where the Kupffer cells were only moderately swollen and prominent.

In brief, the histologic picture of the lymph nodes consisted in a dense proliferation of plasma cells and lymphocytes, a marked infiltration with eosinophils and macrophages, and a marked swelling and proliferation of the reticulum cells with the formation of large atypical mononucleated and multinucleated cells.

In the retropharyngeal and cervical nodes, the addition of tumor cells and necrosis completed the picture of the marked toxic proliferation of the lymph node caused by secondary infection and septicemia.

From the pathologic standpoint, the difficulty of arriving at a definite diagnosis from the biopsy specimens received is apparent. The real difficulty arises in the interpretation of the tremendous response of the plasma cells in association with an atypical proliferation of reticulum cells and their apparent derivatives. From a study of the sections, the transition between mature lymphocytes, irritation forms and typical plasma cells can be readily traced. Similarly, all stages of transition can be found from reticulum cells to the atypical large mononuclear forms and the occasional multinucleated cells (forms which in some instances are indistinguishable from those seen in Hodgkin's disease).

Maximow,[17] in reviewing the relations of the blood cells to connective tissue and endothelium, emphasized the multiple potentialities of the various forms, especially the histiocytes.

Michels [18] published a critical review of the morphogenesis, function and developmental potentialities of the plasma cell. He listed the various possible origins of the plasma cell on the basis of a complete review of the literature, summarizing as follows: (1) connective tissue cells including lymphocytes, fibrocytes, plasmatocytes, resting wandering cells, etc.; (2) emigrant lymphocytes of hematogenic origin; (3) emigrant preexisting tissue lymphocytes, and (4) immature blood cells. As to the function of the plasma cell, he found a variety of theories in the literature, most of which directly or indirectly assumed them to be defensive.

Miller [19] concluded from experimental studies in which he induced the production of large numbers of plasma cells by intraperitoneal injections of tuberculoprotein that the precursor of the plasma cell is a primitive connective tissue cell. He noted that when plasma cells were found, especially in the lymph glands, they were in the connective tissue cords. He concluded that plasma cells constitute a definite entity with a maturation cycle, and that they are stimulated to proliferation by certain toxic agents.

The work of these authors indicates the variety of origins of plasma cells. The findings in this case confirm the assumption that the plasma cell is not of specific origin but is a peculiar form which may arise from mesenchymal cells as a result of some fairly long-continued irritation, usually by a toxin. In this case a streptococcus was isolated from the

17. Maximow, A. A.: Relation of Blood Cells to Connective Tissues and Endothelium, Physiol. Rev. **4**:533, 1924.

18. Michels, N. A.: Plasma Cells: Critical Review of Its Morphogenesis, Function and Developmental Capacity under Normal and Abnormal Conditions, Arch. Path. **11**:775 (May) 1931.

19. Miller, F. R.: Induced Development and Histogenesis of Plasma Cells, J. Exper. Med. **54**:333, 1931.

blood stream and from the cervical and pharyngeal lesions. Miller demonstrated that the intraperitoneal injection of tuberculoprotein would result in a similar development, and undoubtedly a wide variety of bacteria and bacterial products can be incriminated.

Similarly, the occurrence of a marked stimulation of the reticulum cells may be associated with the development of a variety of atypical cells, including the giant cells generally held to be specific for Hodgkin's disease.

While one may be correct in regarding Hodgkin's disease as an entity, its identity must be based not only on the individual cell components of the lesions but more especially on their arrangement and numbers with relation to each other, and the clinical features of the case must be taken into consideration in order to interpret the findings correctly.

SUMMARY

Plasmocellular responses of tissue may accompany infiltrative processes. They may be local or systemic. The granulomas resulting from fairly long-continued sepsis are extremely confusing both in their clinical course and in their microscopic picture. We may well separate this entity under the term of plasma cell granuloma. This response of the lymph glands and tissues is usually the result of a generalized septicemia, which may or may not be associated with a malignant condition. The clinical course is dependent entirely on the etiologic factor. Histologically, the tissues show a dense plasma cell infiltration or proliferation, a marked and frequent bizarre response of the reticulum cells and not infrequently, as is common in chronic lymphadenitis, a more or less eosinophilic response.

ADENOIDS AND IMMUNITY

A FURTHER CONTRIBUTION

HENRY B. LEMERE, M.D.
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The conclusion in my previous paper [1] was that the adenoid is a physiologic lymphatic structure admirably designed to expose the greatest possible lymphatic surface as a barrier to the entrance of nasal infection through the nasopharyngeal mucosa into the general circulation.

In a series of over three hundred children the adenoid was left at the time the tonsils were removed. Daily expulsive blowing exercises were insisted on as after-treatment. Nasal breathing improved; colds and sinus symptoms and acute and chronic aural conditions were less frequent.

The problem of immunity in infections of the upper respiratory tract in children involves so much in its ultimate results that I do not think an apology is necessary for again presenting it for consideration. The end-results of infections of the nasal sinuses involving deeply the mucous membrane and also the bone, in varying degrees of osteomyelitis, and the mastoid involvements which extend to the sigmoid sinus, the petrous tip and the meninges or brain have a beginning. Often, I am convinced, this dates back to residual infection of the head commencing in early life. These terminal changes are less frequent and less violent in so far as one is able to control the prevalent early invasions.

The conception of the therapy of low grade catarrhal sinusitis as being mainly mechanical is borne out by the primary reaction to a cold compared with that to other infections of the upper respiratory tract. In a normal nose this consists of a free mucous discharge and local congestion with only a slight rise of temperature or increase of pulse rate. Other infections of the upper respiratory tract, such as diphtheria, scarlet fever and measles, are accompanied by a profound general reaction and would seem to demand serum therapy. The success of this therapy in diphtheria and its promise in scarlet fever place these infections in a definite class. The indifferent success of biologic therapy in colds indicates that they come under a different therapeutic classification, and that efficiency in treating these infections will come from

1. Lemere, H. B.: Adenoids and Immunity, Am. J. Dis. Child. **43**:1494 (June) 1932.

aiding the natural processes, by leaving the adenoid intact and by assisting in the local cleansing process to the fullest degree.

In combating the harmful effects of infections of the head in children, the prime requisites are efficient nutritional and vitamin feeding, thorough mechanical cleansing, protection from sudden changes in temperature and the preservation of the function of the adenoids.

To give some support to my position in regard to the preservation of the adenoid, I shall quote from the monograph sent to me by Mr. T. B. Layton,[2] of London, England, after I had written my previous article. It is unfortunately not available in this country. The conclusions arrived at by Mr. Layton and myself, independently, closely parallel each other.

Mr. Layton stated in the introduction to his monograph: "In addition to my clinic at Guy's, eight years at the L. C. C. School Clinic of St. George's Dispensary and ten years as Otologist to the Fever Hospitals of London have given me an experience that is, I believe, unusual." I have taken the liberty to parallel several paragraphs from his monograph with those from my earlier paper.[1]

LAYTON.—In all medicine there is no worse term than "adenoids." The term is a vague and indefinite one on the borderline between physiology and pathology . . . and is generally accepted as meaning a definite pathological and clinical "entity." It is assumed that operation is the only means whereby this can be remedied; and the decision to perform this operation is hastily made without that weighing of the pro's and con's that its importance demands.[3]

LEMERE.—"Adenoids" is one of the unfortunate terms of medicine, and even the term "vegetative growths" has been applied to the little structures. These terms seem to preclude the possibility of considering the adenoid organ as a normal physiologic structure.[4]

LAYTON.—For the past twelve and one-half years, after washing them in running water I have examined all masses of adenoids that I have removed. During that period in only three have I seen crypts full of purulent material such as one finds in tonsils, and which we look upon as one of the criteria of their sepsis. The tonsil problem is one of sepsis.[5]

LEMERE.—The tonsil is so constructed that it retains infection; the gross construction of the adenoid, however, is such that, generally speaking, it does not retain infection and is healthy tissue, and the pus seen at times on the surface of the specimen removed comes from the posterior drainage of the nose.[6]

LAYTON.—With regard to the difficulty of breathing through the nose there are two things to consider: first, whether the child has a difficulty in so breathing, and,

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2. Layton, T. B.: On the Conservation of the Lymphoid Tissue of the Upper Respiratory Tract, London, Ash & Co., Ltd., 1932.
3. Layton,[2] p. 5.
4. Lemere,[1] p. 1494.
5. Layton,[2] p. 9.
6. Lemere,[1] p. 1496.

secondly, whether this difficulty is due to an obstruction in the naso-pharynx and not in any other part of the respiratory tract.[7]

LEMERE.—Mouth breathing is frequently caused by nasal infection.[6]

LAYTON.—It is the relation of the amount of lymphoid tissue to the size of the naso-pharynx that is important and not the actual amount of lymphoid tissue present. What you set out to feel is not the soft mass at the top of the naso-pharynx, but the amount of space in front of this between it and the posterior nares.[8]

LEMERE.—While the patient is under anesthesia, before the adenoid is removed, the fingers should be passed into the nasopharynx, not for the purpose of palpation of the adenoid, but rather to determine, by feeling forward to the posterior nares and then backward to the adenoid, the amount of space available for breathing. Even when a large adenoid is present, the space is usually sufficient to allow the finger to move freely forward and backward.[9]

LAYTON.—The treatment is breathing exercises, not operation.[7]

LEMERE.—In cases in which the adenoid is allowed to remain, persistent and adequate attempts should be made to keep the nose clear, and the air douches (blowing exercises) should be continued over a period of months or years.[9]

LAYTON.—Under our present conditions of life no child can escape infection. On many occasions this is of the subclinical type in which the organisms gain access to the nose and throat, but the reaction on the part of the tissue in these regions is not great enough to produce symptoms. On other occasions, however, the reaction is greater and the child is described as having a cold. When this happens the mucous membranes swell, and that which swells most is the lymphoid tissue. This swelling then produces certain symptoms, but as the swelling is of a transient nature it should not be called "adenoid."

To have a certain number of colds is, then, a normal experience in the life of a child. In my opinion it is desirable that they should occur.[10]

LEMERE.—I think that too much value may be attached to the subsidence of the phenomena of colds. If this results from the building up of the general resistance and from proper care and hygiene, the gain is real, but if the bacterial infection occurs and the reaction typical of a cold does not take place because of a fibrous and sclerosed nose or lack of general resistance or because the adenoid is removed, the gain is doubtful. It may even be that the absence of the usual symptoms of a cold indicates a loss in the defense against infection of the upper respiratory tract.[11]

With obstructive breathing, otitis media, progressive deafness and general ill health accompanying conditions of the upper respiratory tract some therapeutic action must be taken.

If it is believed that the adenoid has a definite part to play against bacterial invasion, there should be an effectual program to clear up the nasal infection as far as possible.

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7. Layton,[2] p. 13.
8. Layton,[2] pp. 14 and 15.
9. Lemere,[1] p. 1501.
10. Layton,[2] p. 8.
11. Lemere,[1] p. 1499.

The following is the routine home treatment prescribed for children in my clinic at the Marion Davies Foundation. They are given a Birmingham nasal douche of good quality and this prescription:

	Gm. or Cc.
Sodium biborate	11.6
Sodium bicarbonate	11.6
Phenol, 95%	7.7
Glycerin	31.1
Peppermint water to make	248.8

These written directions are given:

Dilute 1 part medicine with three parts lukewarm water. Throw head way back and allow a little solution to run into the nose, holding the breath. Hold nose and lean head far down. Blow gently at first with both nostrils open. Use in the morning and in the evening at least one half hour before retiring.

The children and parents are taught to hold the breath with the mouth open, while the medicine runs into the nose. The nose is then closed and the head bent far down. Following this there is gentle blowing of the nose and clearing of the throat for three or four minutes. The nose and throat now being clear, exercises are given consisting of deep inspiration through the mouth and forceful expiration through the nose, both nostrils being left free. This blowing exercise is repeated from about eight to twelve times.

In theory the recent work on ciliary action of the nasal mucosa may be urged as an argument against solutions, but in practice I find that they remove the thick, infected mucus and stimulate the secretion of normal mucus. This is simply one method. Campbell and others employ suction cleansing, which I also use when it is practicable.[12]

If the adenoid is removed and no mechanical cleansing is instituted, nasal obstruction may persist; a diagnosis is then erroneously made of regrowth of the adenoid, and another operation is performed. At times I have known this to be repeated two or three times. Usually, from necessity, no effort is made to see the adenoid or to find how much it is actually obstructing breathing, nor is any determined effort made to keep the nose clear over a period of weeks to see if the obstruction disappears. Consequently, the diagnosis of adenoids as generally made would better be simply nasal obstruction.

In my work in the dispensary at the Marion Davies Foundation I have the advantage of a good social service department and a limited district, so that the follow-up of the ordinary dispensary care is better than usual. As soon as the children are progressing satisfactorily they are instructed to continue their nasal hygiene and to return in three months. The effect of this method on symptoms commonly attributed to adenoids but in fact due to nasal infection has been as follows:

12. Campbell, E. H.: Tr. Am. Laryng., Rhin. & Otol. Soc., 1932, p. 163.

RESULTS OF TREATMENT

Mouth Breathing.—Improvement occurred in all patients with only one exception. In this case, after breathing exercises and cleansing had been carried on for weeks, the adenoids were finally removed as there was still obstruction. Layton thinks that the open mouth does not necessarily mean mouth breathing. In experimenting on myself I found that when breathing with the mouth open passage of air occurs simultaneously back and forth through the nose. Mouth breathing generally results not from the adenoids but from nasal obstruction; it may become habitual and be due partly to laxity of the muscles which hold up the mandible. Some of these children have to be trained to hold the jaw in its proper place. This, however, should be taught after the patient can breathe easily and without sound through the nose.

Acute Otitis Media.—Very few patients in this series have returned with acute suppurative otitis media, either discharging or requiring paracentesis. This at least shows that diseases of the middle ear are not more prevalent when the adenoid is left. It is possible that they are less frequent. During the recent epidemic of measles not one child in whom the adenoid was left had acute suppurative otitis media.

Catarrhal Deafness.—These patients were treated by politzerization, but only after they had practiced nasal hygiene for some time and the nose, pharynx and tubal openings were free from mucopus. They rapidly improved to the point where they heard so well that they had no trouble in their classes at school, and this improvement persisted. When mechanical cleansing of the nose and pharynx is not thoroughly carried out politzerization results in catarrhal infection of the middle ear and, together with the bad effects of retraction of the drum, leads to progressive deafness.

There may be several factors producing obstruction of the eustachian tube. Heavy mucus over its orifice effectually prevents the air from entering the tube. When the mucus disappears the tubes remain open and are not obstructed by the rugae of the adenoid.

Colds.—Colds were still fairly frequent, but the exercises and cleansing urged particularly after the acute symptoms of the cold had passed caused a return to normal health more rapidly. The general appearance of those children who had swollen turbinates and considerable nasal discharge during a cold was often much better than that of others in whom the nose was patulous during a cold with scant discharge, mainly posteriorly, producing coating of the pharynx, redness and swelling of the lateral pharyngeal angles or a granular condition of the posterior pharyngeal wall.

Broad Nasal Bridge.—When catarrhal conditions have remained for some time a broad nasal bridge sometimes results. It comes from obstruction in the middle fossa and ethmoidal region and not, as

popularly supposed, from adenoids. In cases followed for over two years the parents have often commented on the diminution of this swelling.

Disease of the Sinuses.—All patients coming into my clinic were free from acute empyema. The maxillary sinus often appeared cloudy on roentgenograms and mucopus could be washed out in an occasional case, but in this series I saw only two patients who I thought would improve more rapidly with operation than with the use of nasal hygiene. A posterior pharynx that is coated with mucopus or glazed or that has red lateral streaks or red follicles indicates drainage posteriorly from the nose and its sinuses.

Much has been done in the last two decades in the treatment and understanding of catarrhal infections of the upper respiratory tract. The thought and care given to infant feeding with sufficient nutritional and vitamin content must produce increased resistance to infections of the upper respiratory tract. Parents can be taught to desist from spraying their children with bacteria when they talk directly to them. The value of sunlight is generally recognized. The effect of variation in temperature is counteracted by better hygiene and more rational clothing.

Biochemistry is furnishing basic truths which it will be possible to use in a practical manner. I am satisfied that the function of the adenoid in protection against infections of the nose and nasal sinuses will before long be recognized and that the adenoid not only will be allowed to remain but will be aided in its action by thorough cleansing of the nose and throat.

The outlook is hopeful that with the coming generation there will be fewer serious end-results of infections of the upper respiratory tract.

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UNILATERAL MALFORMATION OF THE EAR ASSOCIATED WITH CYCLOPIA

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Friedberg Fellow in Otolaryngology, University of Chicago
CHICAGO

REPORT OF CASE

History.—Cy., a full term baby girl, was delivered on Feb. 24, 1932, by Dr. W. Serbin at the Wesley Hospital. During vaginal examination, prior to delivery, it was found impossible to palpate the ear with the examining finger. This was explained, after birth, by finding practical absence of the left ear. Because of the hydrocephalus craniotomy was necessary for delivery. The obstetric phase of this case has been reported in the *American Journal of Obstetrics and Gynecology*.[1] The baby's mother was a primipara, 35 years of age. The general history of both the father and the mother was unimportant; no venereal infection was reported.

Necropsy.—A general postmortem examination revealed hydrocephalus, a rudimentary brain, cyclopia, absence of the left external ear, absence of the external nose and premaxilla and a complete cleft of the upper lip (fig. 1). Examination of the organs showed hypoplasia of the thyroid and suprarenal, retarded development of the liver, lungs and suprarenal cortex and fibrosis of the thyroid and pancreas, while the thymus, spleen, heart and urogenital organs were apparently normal.

More detailed gross examination of the brain showed the following, according to Dr. G. Bartelmez of the University of Chicago: "The base of the brain revealed no evidence of the optic chiasm or of the third nerves. The fourth nerves appeared normal on each side. The fifth was present and normal on the right side, but on the left only a few filaments connected the nerve with the brain. The sixth nerves were apparently normal. It was also possible to identify the eighth and the intermedius on each side. However, while the motor seventh was present on the right side it could not be found on the left. The macerated condition of the brain made it impossible to identify accurately the ninth to the twelfth nerves, especially on the left side. On the other hand, nothing was seen which indicated any abnormality. The dorsal surface of the brain showed the cerebellum and a rather distorted midbrain. The cerebral hemispheres were represented by a small shapeless mass of tissue connected to the brain stem by a slender stalk. It was impossible to recognize definite structures. The mass was flattened as though compressed by the hydrocepalic fluid."

The base of the skull showed absence of the olfactory bulbs and optic chiasm. A single nerve was seen to course anteriorly where the chiasm should be. This was later followed to the right rudimentary eye. The third nerves were not identified. The remaining cranial nerves seemed present except that less nerve tissue was seen to enter the internal auditory meatus on the left side (absence of the seventh nerve). No definite infundibulum or pituitary body was seen. The base of the skull was deficient anteriorly where the ethmoid bones are normally situated. The eyes were

1. Hagens, E. W.: Am. J. Obst. & Gynec. **25**:758 (May) 1933.

represented by two rudimentary structures, one larger than the other, located in the site of the normal ethmoid sinuses and nose. The left eye was soft and measured about 1.5 cm. in diameter. It contained degenerated vitreous, and was lined with retina but not with choroid. Anteriorly an opaque cornea seemed represented, with a small spot of pigment. No nerve could be traced to this eye. The right eye was rudimentary, being only about 4 by 7 mm. It had a bluish cast, and

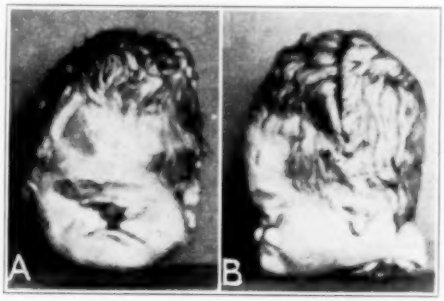

Fig. 1.—*A*, cyclopia, absence of the nose, and cleft of the upper lip. *B*, left side of the head, showing absence of the ear except for the lobule.

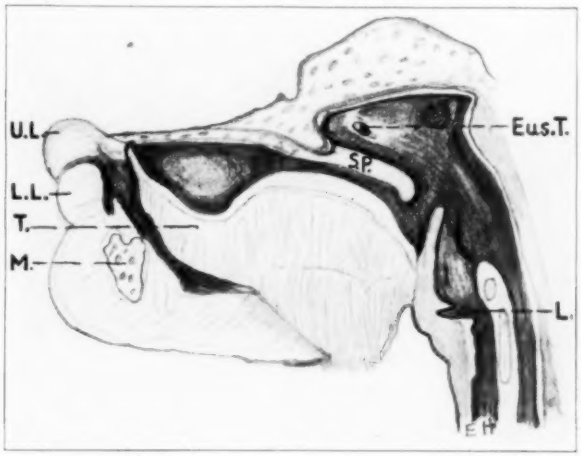

Fig. 2.—Right side of the head after a median section: *U. L.*, upper lip; *L. L.*, lower lip; *T*, tongue; *M*, mandible; *S. P.*, soft palate; *Eus. T.*, eustachian tube, and *L*, larynx.

contained choroid and shrunken retina. The nerve mentioned in the description of the base of the skull led into this eye. The external ocular muscles and the nerves to them could not be absolutely identified; however, about four or five muscles altogether were seen. The two eyeballs were definitely separate structures, although closely attached by connective tissue. The eyebrows were separate. The upper lids were fused into one, while the lower lids were present only in their lateral halves, and on the left side the eyeball was fused to the margin of the lid.

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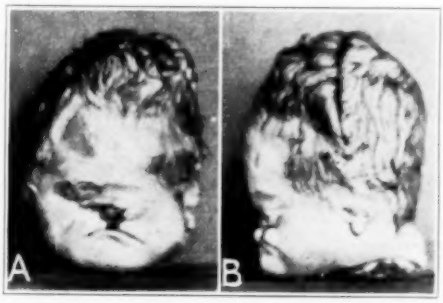

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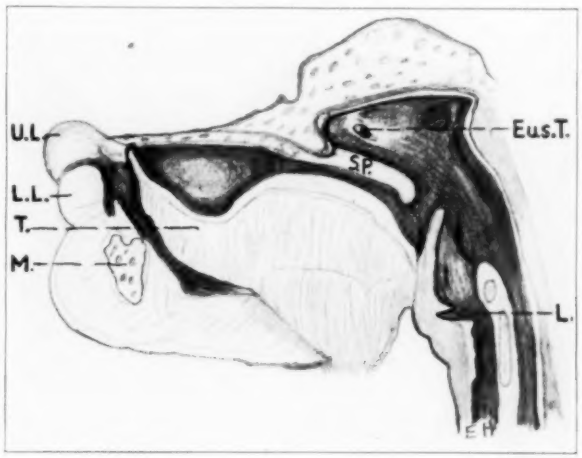


Fig. 2.—Right side of the head after a median section: *U. L.*, upper lip; *L. L.*, lower lip; *T*, tongue; *M*, mandible; *S. P.*, soft palate; *Eus. T.*, eustachian tube, and *L*, larynx.

contained choroid and shrunken retina. The nerve mentioned in the description of the base of the skull led into this eye. The external ocular muscles and the nerves to them could not be absolutely identified; however, about four or five muscles altogether were seen. The two eyeballs were definitely separate structures, although closely attached by connective tissue. The eyebrows were separate. The upper lids were fused into one, while the lower lids were present only in their lateral halves, and on the left side the eyeball was fused to the margin of the lid.

After removal of the eyes the head was sectioned in the median plane. Examination showed complete absence of the nose, the nasopharynx ending blindly in the region of the eustachian tubes (fig. 2). The tubes were normal, as were the rest of the pharynx, the oropharynx, the tongue, the soft palate, the larynx and the hypopharynx. The hard palate was intact, but the absence of the premaxilla and upper lip produced a cleft in the anterior part of the mouth. The following bones were apparently absent: the nasal, ethmoid, lacrimal, palatine, premaxilla, inferior turbinal and vomer.

Ears: On gross examination, the right ear and temporal bone were normal. The left auricle was practically absent, being represented by the pedunculated lobule. At its base was a small mass of cartilage. No external auditory canal was present, and the region of the membrana tympani was filled in by bone and connective

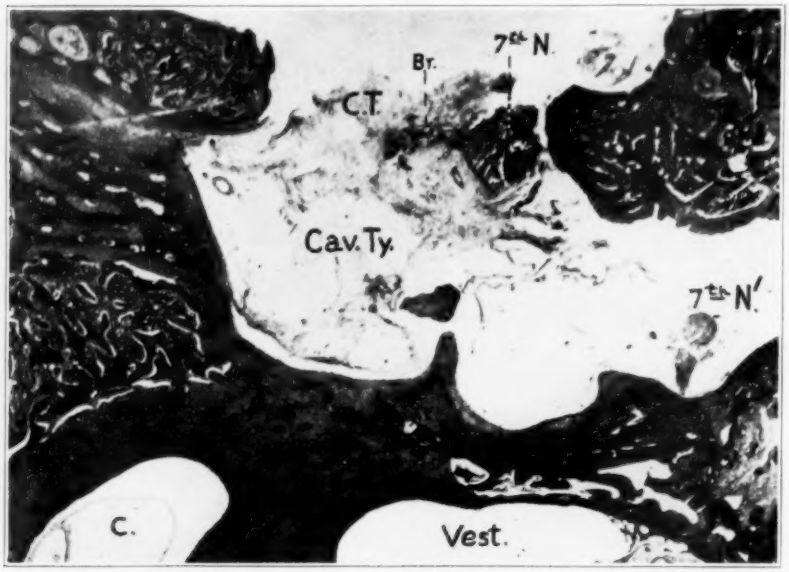


Fig. 3.—Left, horizontal section: *7th N.*, seventh nerve making its exit from the middle ear cavity (*Cav. Ty.*) through dense connective tissue (*C. T.*) and sending a small branch (*Br.*) posteriorly; *7th N.*, seventh nerve in the region of the geniculate ganglion; *C.*, a semicircular canal, and *Vest.*, vestibule; × 12.5.

tissue. Instead of making its usual knee, the internal carotid artery entered the petrous bone on its lateral surface and then coursed directly forward. The remainder of the petrous bone appeared normal, except that there seemed to be a lack of fulness in the region of the tegmen tympani.

Microscopic examination of the sections of the right or normal ear showed a normal membrana tympani, ossicles and tensor tympani and stapedius muscles. The mucous membrane of the cavum tympani was of the embryonic type. In the tympanic cavity were collections of polymorphonuclear and red blood cells. The region of the antrum was filled in with embryonic connective tissue containing numerous blood vessels. The cochlea, saccule, utricle, cristae and seventh, eighth, chorda tympani and petrosal nerves showed only postmortem changes. A definite focus of cartilage was noted lying between the cochlea and the vestibule.

The left or deformed ear revealed the absence of the membrana tympani, this region being filled in by bone and coarse connective tissue. The malleus and incus were absent (fig. 3). The stapes, while present, did not have the usual form (fig. 5). The tensor tympani muscle was represented by a structure resembling muscle under the low power microscope, but under oil immersion muscle fibers could not be demonstrated. The stapedius muscle seemed normal. The cavum tympani was definitely smaller than that on the right, or normal, side. At the level of the stapes (fig. 5) it was 3 mm. lateromedially by 6 mm. anteroposteriorly instead of 7 by 10 mm. as on the right side. At the level of, and opposite to, the round window (fig. 6) it was 2 mm. lateromedially instead of 5 mm. The cavum

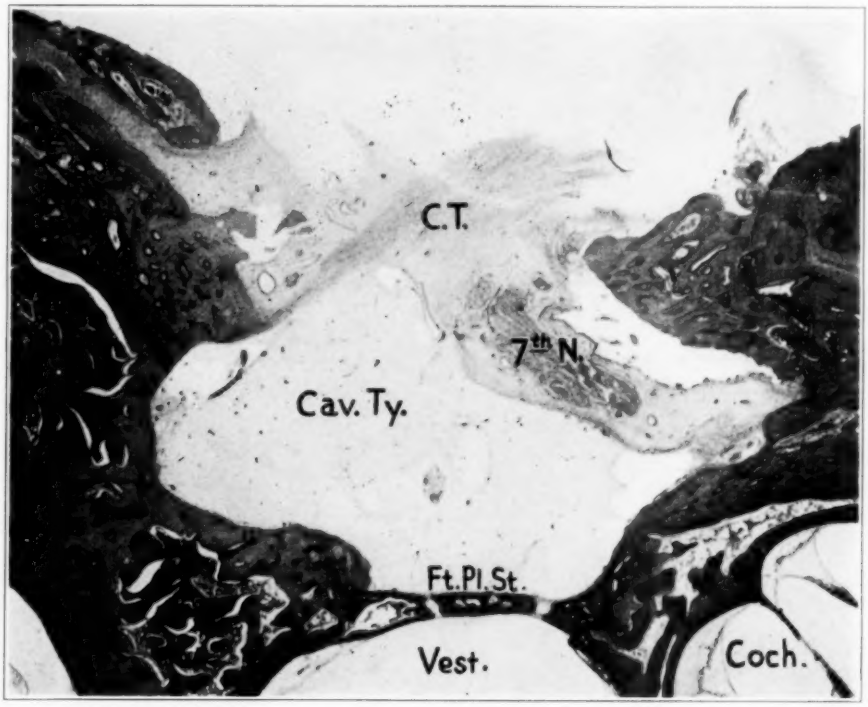


Fig. 4.—Left, horizontal section: *C. T.*, dense connective tissue filling in the area where the membrana tympani should have been; *7th N.*, seventh nerve coursing abnormally across the cavum tympani (*Cav. Ty.*); *Ft. Pl. ST.*, footplate of the stapes; *Coch.*, cochlea, and *Vest.*, vestibule; × 12.5.

tympani contained embryonic connective tissue for the most part, with coarse connective tissue in places, especially laterally. The entodermal epithelium extended only as far back as the apex of the cochlea (fig. 6). This portion was continuous anteriorly with the eustachian tube. A cavity corresponding to an antrum and filled with embryonic connective tissue was seen, but it did not join the cavum tympani in the usual manner. The cochlea, saccule, utricle, cristae and both branches of the eighth nerve showed only postmortem changes. The seventh nerve appeared to be absent in the internal auditory meatus. Between the cochlea and the vestibule its stump was found, extending to the region of the geniculate ganglion. Here,

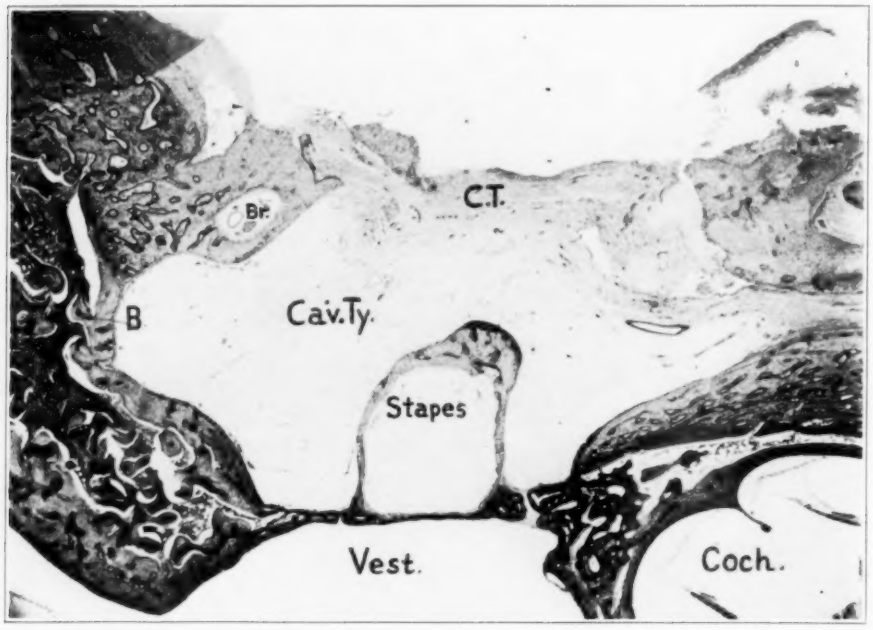


Fig. 5.—Left, horizontal section: *C. T.*, dense connective tissue filling in the area where the membrana tympani should have been; *Br.*, tiny branch of the seventh nerve seen in figure 3; *B*, bay formation, which on the right (normal) side is the site of the fallopian canal and contains the seventh nerve; *Cav. Ty.*, cavum tympani filled with embryonic connective tissue; *Vest.*, vestibule, and *Coch.*, cochlea; × 12.5.

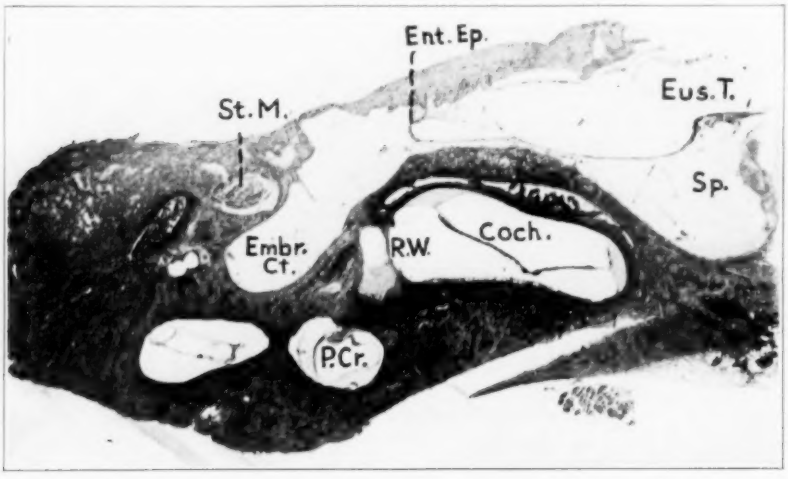


Fig. 6.—Left, horizontal section: *Ent. Ep.*, entodermal epithelium which ends at this point, but is continuous anteriorly with the eustachian tube (*Eus. T.*); *St. M.*, stapedius muscle; *Embr. Ct.*, embryonic connective tissue in the cavum tympani; *R. W.*, round window; *Coch.*, cochlea; *Sp.*, space which is filled with embryonic connective tissue, but which should contain the internal carotid artery, and *P. Cr.*, posterior crista; × 5.

instead of following the usual fallopian canal the nerve extended directly across the cavum tympani, supported by coarse connective tissue (fig. 4). At the lateral boundary of the tympanic cavity a small offshoot of the nerve extended posteriorly (fig. 3). The latter gradually worked its way posteriorly and, becoming encased in a bony canal, followed somewhat the course of the usual fourth portion of the seventh nerve (fig. 5). A small part of it was seen to supply the stapedius muscle, while the rest of it became external as does the normal seventh nerve at the stylomastoid foramen. The main part of the nerve, after crossing the tympanic cavity, became external by penetrating the connective tissue which replaced the region of the membrana tympani. The space which on the right side was occupied by the fascial nerve in its fourth portion was on this side represented by a bay formation in the bone (fig. 4). No chorda tympani nerve was seen, but nerve tracts were noted extending anteriorly from the geniculate ganglion as on the other side (petrosal nerves). The ganglion was surrounded by connective tissue and lay abnormally free in the cavum tympani. The round window was found normal (fig. 6). The space occupied by the knee of the internal carotid artery on the right side was represented on this side, but was filled in with embryonic connective tissue (fig. 6).

COMMENT

The infant described presented multiple deformities, i. e., hydrocephalus, cyclocephalus, cleft lip and palate, malformation of the left ear and abnormalities of several of the cranial nerves. Cyclopia has been produced experimentally in several different ways. Early maldevelopment of the forebrain and other abnormalities in this region would result in cyclocephalus. A blockage between the foramen of Monro and the third ventricle would lead to hydrocephalus. Absence of the first and third nerves and abnormality of the optic nerves would also be expected. Absence of the premaxilla and a cleft of the upper lip would seem to be a separate deformity, and it is difficult to state whether such a condition results from a primary aplasia or from a secondary degeneration. Malformations of the auricle, external auditory canal and cavum tympani are frequently found to be related. In the majority of such cases the inner ear is normal, as in the present instance. The absence of the malleus and incus, with the stapes present, is in conformity with observations in similar cases. The tensor tympani muscle could not be recognized histologically as muscle tissue, whereas the stapedius muscle was normal. One wonders whether the change in the tensor muscle was not related to the absence of the malleus.

The pneumatization of the cavum tympani by the entodermal epithelium of the first pouch stopped opposite the apex of the cochlea. In a case previously reported [2] a similar situation was found. However, in that case thymus tissue was also found at this point. It was stated that perhaps the thymus tissue had something to do with the lack of

2. Hagens, E. W.: Malformation of the Auditory Apparatus in the New-Born Associated with Ectopic Thymus, Arch. Otolaryng. **15**:671 (May) 1932.

further extension of the entodermal epithelium. However, the present case shows the same phenomenon without the presence of thymus tissue.

The course of the seventh nerve through the cavum tympani is of interest. In relatively few cases has the nerve been found to follow this course. More often it is normal or thinned, but follows its usual direction through the facial canal. The geniculate ganglion and petrosal nerves were present, but the chorda tympani nerve was absent. One may wonder what would happen to the facial nerve in such a case if the child were living and an otitis media developed, or if the surgeon were forced to operate to produce drainage. In the present case the facial nerve did not appear to be connected to the brain. Apparently some process caused degeneration of the central connection with the brain. The junction of the fifth nerve on the same side (left) was almost destroyed. The basal nuclei could not be studied because of the extremely poor preservation of the brain. Roentgenograms of the base of the skull and lower jaw revealed no bony abnormalities. The coexistence of the malformation of the ear with the other deformities is of interest. Apparently such a condition is of rare occurrence. One may speculate whether at the time the forebrain and optic vesicles were being affected the region of the hyomandibular pouch was not also being involved.

CONCLUSION

A case of malformation of the ear associated with hydrocephalus and cyclocephalus is presented. The left auricle was represented only by the lobule; the external auditory canal was absent, as were also the membrana tympani, malleus and incus. The stapes and stapedius muscle were present. The inner ear was normally formed. The facial nerve was not connected with the brain, but its peripheral part was present, crossing, however, directly through the cavum tympani. The latter was smaller than normal and was for the most part filled with embryonic connective tissue. The entodermal epithelium of the first pouch extended only as far posteriorly as the apex of the cochlea.

Clinical Notes

SINUSITIS AND NEURALGIA

H. Garber, M.D., Montreal, Canada

Neuralgic pains frequently occur during the course of a chronic sinusitis. As these are in the nature of referred pains, being felt at points distant from the pathologic site, it is likely that they are due to the involvement of some nerve. However, attempts that have been made to designate particular and rarer forms of these neuralgias as syndromes linked with some specific nerve structure have not been entirely convincing. Thus Sluder's conception of a sphenopalatine syndrome and the designation of this same syndrome as vidian neuralgia are based on neurologic interpretations which cannot be considered perfect.

Sluder's[1] own description of his syndrome is as follows: "Coryza followed by pain starting at the root of the nose, in and about the eye, involving the upper jaw and teeth, sometimes also the lower jaw and teeth, extending backwards to the temple and about the zygoma to the ear, emphasized at the mastoid, but always severest at a point five centimeters back of that; thence reaching backwards by way of the occiput and neck; it may extend to the shoulder, the arm and even to the fingertips."

Sluder linked this syndrome with an involvement of the sphenopalatine ganglion and divided the syndrome into two parts: the anterior pains involving the face and eye, traveling along the nerves that emerge from this ganglion, and the posterior pains of the occiput, neck and shoulder, traveling along the vidian nerve, the sympathetic plexus on the internal carotid artery, the superior cervical sympathetic ganglion and thence along the cervical nerves.

H. H. Vail,[2] who considers that it is the vidian nerve and not the sphenopalatine ganglion which is involved in the inflammatory process in these cases on account of the close association of that nerve with the sphenoid sinus and the fact that he was able to demonstrate a sphenoiditis in a number of cases presenting the symptoms of Sluder's syndrome, offers an essentially identical neurologic interpretation. The supposed course of the pain impulses is the same except that the starting point is in the vidian nerve and the pains to the ear travel by way of the anastomotic branch of the facial and the tympanic plexus. Pains sometimes felt in the throat in this syndrome travel from the tympanic plexus along the tympanic branch of the glossopharyngeal nerve.

An essential part of both explanations is that the pains of the neck and shoulder are carried by the sympathetic system. This assumption seems unwarranted, since it is doubtful if sympathetic fibers ever act as pain conductors. Evidence based on

1. Sluder, G.: Concerning Some Headaches and Eye Disorders of Nasal Origin, St. Louis, C. V. Mosby Company, 1918.

2. Vail, H. H.: Vidian Neuralgia, Ann. Otol., Rhin. & Laryng. **41**:837 (Sept.) 1932.

the type of cells found within the sympathetic ganglions and on the character of the nerve endings of sympathetic fibers has led many physiologists to the belief that no sensory fibers are present in the sympathetic nerves. De Castro,[3] in Penfield's "Cytology of the Nervous System," stated: "The existence of sympathetic sensory neurons in the ganglia of the autonomic nervous system is very unlikely." Ranson,[4] in his "Anatomy of the Nervous System," stated: "We have shown that there are probably no association or sensory neurons in the sympathetic ganglion and if this be true, there are no axons arising from such cells in the sympathetic nerve trunks and plexuses." Even the afferent fibers contained in the sympathetic nerves, according to Ranson, do not convey impulses which create sensations rising above the subconscious level.

That the vidian nerve does not act as a conductor of pain impulses can be proved clinically by the fact that a blocking of the gasserian ganglion gives complete anesthesia over the area supplied jointly by the fifth and vidian nerves, viz., the lower part of the nose, maxillary sinus and palate.[5] If the vidian nerve contained pain fibers, this nerve as well as the fifth would have to be blocked in order to obtain anesthesia of these parts.

It follows that both components of the vidian nerve are free from pain fibers, the great superficial petrosal as well as the deep petrosal (this also rules out a suggestion recently made,[6] linking the great superficial petrosal with the pains of Gradenigo's syndrome). The function of the great superficial petrosal is still uncertain. It is probable that this nerve is the cranial autonomic component of the sphenopalatine ganglion, of which the deep petrosal is the thoracolumbar sympathetic component. This is in line with the general plan of sympathetic innervation in the head.

A better explanation of the posterior pains of Sluder's syndrome, that is, the pains of the neck and shoulder, is afforded if one assumes that it is the maxillary nerve, and not the vidian, which is the starting point of these pains. The maxillary nerve is in just as close proximity to the sphenoid sinus as is the vidian. From the maxillary nerve the impulses may be assumed to reach the neck and shoulder along the descending or spinal tract of the trigeminal nerve, which extends downward to the level of the second cervical nerve. The assumption that the maxillary nerve is the one involved in these cases also affords a better explanation of the anterior pains, since the fibers from the sphenopalatine ganglion do not reach the skin. The fact that the maxillary is a purely sensory nerve and a very large one adds to the likelihood that this interpretation is correct.

It seems unreasonable to suppose that the involvement of a small nerve could give rise to pains, either direct or referred, which are felt over a large area. This follows from a consideration of the mechanism of referred pain. The fact that referred pains are so often observed when some nerve is known to be involved in

3. de Castro, in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932.

4. Ranson, S. W.: Anatomy of the Nervous System, Philadelphia, W. B. Saunders Company, 1927, p. 347.

5. de Takáts, G.: Local Anesthesia, Philadelphia, W. B. Saunders Company, 1928, p. 52.

6. Vail, H. H.: Vidian Neuralgia With Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Apex, Arch. Otolaryng. **17**:212 (Feb.) 1933.

a disease process, and the frequency with which referred pains are set up when a nerve is struck by the needle in the course of procedures for nerve block, would lead one to infer that referred pains, whenever they occur, are due to the involvement of some nerve. Two explanations at once present themselves. The first is that the stimuli are of greater intensity when applied to a nerve fiber in the middle of its course than when applied to its receptor. This is ruled out by the "all or nothing law," which is to the effect that a nerve fiber is either stimulated or not stimulated, and that differences of intensity do not matter. The second explanation appears more likely. When a nerve reaches the periphery, its fibers spread out to cover a more or less extensive area of skin. In the ordinary course of events only a few axons are activated by a stimulus applied to the skin, since the stimulus is not likely to cover the entire area of the skin supplied by the nerve. When the nerve itself, however, is involved in a disease process, or is stimulated artificially by a needle, a large number of fibers are activated at the same time, and there is an overflow of stimuli to fibers of other nerves which happen to make connections with those fibers. This view allows one to regard referred pains quantitatively and compels one to conclude that pains felt over an extensive area of the body, whether they are direct or referred, must be due to involvement of a large nerve trunk.

A further consideration of the mechanism of referred pain leads one to assume that it occurs in the following way: In those cases in which a stimulus applied to one branch of a nerve is felt as though it came from the receptors of another branch, the stimulus was transferred from the fiber to which it was applied to a second fiber at a point where the two happen to come in contact, and the impulse continued along the second fiber in a central direction only and was interpreted by the brain as coming from the nerve endings of the second fiber, since the brain can interpret a stimulus applied anywhere along the course of a neuron only as though it originated from that neuron's receptor. Thus there was no actual impulse from the point of contact to the receptor; the impulse was present only in that portion of the nerve fiber extending from the point of contact to the brain. Any explanation in which it is necessary to assume a peripheral course for the pain impulse must be rejected, since such a course would be in violation of the law of dynamic polarity. Thus Vail's assumption that the pains in the throat are felt there as the result of a stimulus applied to the vidian nerve, traveling thence along the greater superficial petrosal, the geniculate ganglion, the anastomotic branch of the facial, the tympanic plexus and the tympanic branch of the glossopharyngeal, indicates a peripheral course for the impulse from the geniculate ganglion to the tympanic plexus, in order that this impulse may be transferred to the glossopharyngeal. In the case of the pains in the ear this objection does not hold, since the impulse can be transferred from the greater superficial petrosal to the fibers of the anastomotic branch of the facial nerve within the geniculate ganglion, and thence travel centrally along the facial nerve.

These considerations lead to the conclusion that, having regard for the functional composition of the nerves under consideration, the direction of the pain impulses and the size of the nerve structures, the sphenopalatine ganglion and the vidian nerve should not be considered as having anything to do with the neuralgic pains of Sluder's syndrome, and that a better explanation is afforded by regarding the maxillary nerve as the one involved.

It goes without saying that a correct neurologic interpretation is necessary before any attempt to relieve pain by nerve block is contemplated.

RUBBER BUNDLE TYPE OF MASTOID DRAINAGE

CARLETON WILLIAM BULLARD, M.D., WASHINGTON, D. C.

For many years medical literature has offered various means of mastoid drainage. Although the postoperative success of mastoidectomy is not due entirely to the type or method of draining the wound, it is an accepted fact that this plays an important part in the convalescence of the patient. Without adequate drainage many complications can occur. The most significant of these is continued aural discharge, which is due usually to inadequate drainage of the antral region. It is my purpose in this paper to present briefly a method which I believe allows continuous and adequate drainage of the antral region.

It has been my practice to use no packing in the mastoid cavity after a complete exenteration of the mastoid cells. Drainage was maintained by a single wick of gauze extending from the antrum along the floor of the cavity and through the lower end of the skin incision. This small wick was partially removed each day after the operation. I have discontinued the use of the single strand type of drain because a partial withdrawal of this type of drain prevented the deepest point, namely, the antrum, from having any means of drainage. Occasionally this prevented an early dry ear. Therefore, any type of drain which will allow the size of the drain to be decreased without sacrificing the depth of drainage is of advantage.

To accomplish this I have used what I call a rubber bundle type of drainage. This consists of six or eight strands of rubber, about 4 mm. wide (one-eighth inch). The standard size no. 33 (Eberhard Faber) rubber band with the ends removed is preferred. One at a time these are placed in, or as near as possible to, the antrum, and lie on the floor of the bony cavity, emerging from the lower part of the wound for about 2 cm. (three-fourths inch). This allows any type of closure of the wound. It can be seen readily that daily removal of one or more of these strands decreases the size of the drain without interrupting its extent or depth, thus allowing antral drainage as long as is necessary. In this method of drainage I prefer rubber bands of the size mentioned because they allow free removal of one or more strands without displacing or removing the others. Strands of thin rubber dam were unsuccessful, for, on account of their flexibility, the removal of one strand displaced the others. The rigidity of the no. 33 band allows the position of each strand to be maintained during dressings or withdrawal. Another important advantage that this method offers is the freedom from pain on removal of this type of drain as compared with any other procedure. The rubber bundle method has been used by me in mastoidectomies on infants, children and adults, and has served its full purpose successfully in each case.

CONCLUSIONS

1. The single strand type of drain sacrifices depth of drainage even by partial withdrawal.

2. The benefit of continued antral drainage is lost with any single strand type of drain.

3. The rubber bundle type of drain maintains depth of drainage even while it is being reduced in size.

4. It allows removal with little or no discomfort to the patient.

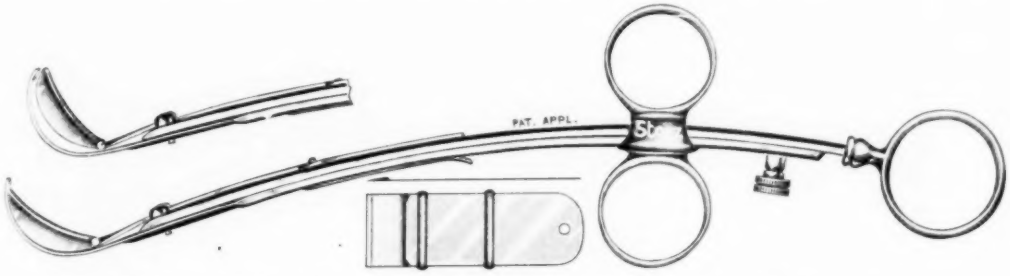
1726 Eye Street, N. W.

A NEW ADENOID INSTRUMENT

DAVID R. HIGBEE, M.D., SAN DIEGO, CALIF.

The adenoid operation, however simple and easily done, is compromised by the rigid type of instrument. Experience shows that clean removal of adenoid tissue is not easily accomplished, as evidenced by the frequency of regrowth after operation.

The adenoid is superimposed on unyielding bony tissue which has no constant contour or angle. A rigid instrument may be manipulated, after being put in position, to include the maximum amount of tissue for each person, but such adjustment is always limited by the spread between the teeth of an open mouth and the fact that neither the instrument nor the posterior wall will yield to pressure.



The upper illustration shows the instrument flexed; the lower illustration, its natural appearance.

The instrument presented here is flexible. When pressed forward, it conforms to the angle presented in the individual patient. Ordinarily pressure in this direction would approximate the lower pole of the adenoid and heel of the fenestrum more closely but also separate the upper pole from the toe of the fenestrum. In this instrument, however, this has been overcome by a difference in tension between the spring of the fenestrum and the blade so that the entire adenoid from base to tip is removed completely.

The blade is furthermore reinforced at the cutting edge to prevent its bowing at the center, so that the adenoid is always cut through cleanly.

When removed, the adenoid appears symmetrical; it shows three distinct lobes and a smooth posterior surface resembling a fibrous tissue capsule.

The instrument is manufactured by the Storz Instrument Company of Indianapolis.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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ACUTE AND CHRONIC OTITIS MEDIA, SINUS THROMBOSIS AND PÉTROSAL PYRAMID SUPPURATION

SAMUEL J. KOPETZKY, M.D.
NEW YORK

My survey this year reveals that most of the recorded observations are concerned with clinical studies. It is apparent from these surveys, which I have conducted for a number of years, that a clearer comprehension of surgical pathology is generally held. The knowledge that the lesion is profoundly influenced by the tissue on which it develops has been won, and the larger part of the studies published are concerned with the application of surgery to these recently acquired fundamentals.

PROBLEMS CONCERNED WITH ACUTE LESIONS

Pneumatization.—Everywhere the question of pneumatization of the temporal bone is being studied. The implications derived from knowledge gained of the structure of the bone on which the pathologic lesion builds itself are becoming clearer. No one even casually reading through current literature can fail to be impressed with the evolution of ideas which the work of Wittmaack has made possible, and the changes in pathologic conceptions have led and are leading to a more rational therapy. It would be far beyond the scope of so limited a paper as this review to attempt discussion of the many details involved in the question; suffice it to indicate outstanding path-pointing studies.

Thus Kriegsman [1] tried to find an answer to the problem of disturbed pneumatization in patients who were otologically normal but who had unimpeachable histories of otitic infections at an early age, though not in the very first weeks or months of life. Kriegsman's studies were made roentgenologically. He divided his material into three groups.

In group I were patients who had suffered an otitic infection at the age of 1 year and since then had been well as far as the ear was con-

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From the Otolaryngological Department of Beth Israel Hospital.

1. Kriegsman: Röntgenologische Untersuchungen an Warzenfortsätzen nach durchgemachter Otitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:259, 1931.

cerned. In this group there were 17 cases. Of these only 3 showed evidence of disturbed pneumatization. The rest gave negative findings. From this Kriegsman deduces that a strong and healthy pneumatization, once started on its career toward producing a normal pneumatized mastoid process, goes on even though there occurs during the first year of life an attack of middle ear infection which heals. The observation coincides with the opinion of Wittmaack, who has shown that a mild intercurrent otitis media which runs its course and clears up will not interrupt the evolution of normal pneumatization.

In Kriegsman's group II were patients who suffered an attack of otitis media during the second year of life. These were mild cases which cleared up uneventfully, although a few of them showed recurrent earache. Fully evolved purulence of the mastoid process never developed. There were some evidences of disturbed pneumatization in this group.

In group III the patients suffered from an attack of otitis media after 4 years of age. In these cases pneumatization was mostly completed, as far as the mastoid process was concerned, prior to the onset of the otitic infections. There were 40 cases in this group. Of these 22 evidenced interference with pneumatization. In 10 there was only a slight abnormality: Eight cases showed considerable change in the pneumatization.

Kriegsman concludes his work with the observation that an attack of acute otitis, even with a perforation of the membrana tympani, when it occurs in early life does not necessarily disturb pneumatization; nor do repeated attacks of mild infection in early life result in the formation of compact bone.

Steurer,[2] discussing the factors which disturb pneumatization, in the main agrees with the findings recorded by Kriegsman. He adds the observation that the structural changes are apt to be similar in both ears. He examined 833 patients, and reports that he found unilateral changes in pneumatization in only 83.

An interesting observation was made by Leicher,[3] who examined a pair of identical twins, one of whom, at the age of 4 months, suffered an attack of purulent otitis media which ran its course and healed without operation. In this patient he found a marked change in pneumatization in the affected ear. The opposite mastoid process, as well as the mastoids of the twin, was normally pneumatized.

Every one has held the idea that once the cellular structure of the mastoid process is exenterated by surgical means, cellular replacement cannot happen. Nevertheless, the report of Voss[3] contradicts this. He operated on a child during the first year of its life, performing what

2. Steurer: Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:113, 1931.

3. In discussion of Steurer.[2]

he terms an antrotomy. When the patient was 9 years of age Voss again operated on the same ear and found complete pneumatization, which he reports as being so extensive that the cells extended into the squama and zygoma and even backward toward the occipital bone. This report becomes increasingly important to those who, for reasons which a patient presents, find it necessary to reoperate. Cellular elements found in the area of the previous operation should not be looked on as "rests" which were overlooked at the original surgical procedure, but rather as the result of a subsequent evolution of cells from elements and factors all of which are not yet comprehended and the genesis of which is not yet clear.

Loebell [4] also studied the mastoid process roentgenologically. His cases also represent patients who had been subjected to operative measures during the evolutionary period of pneumatization. His studies comprise 21 patients. Two thirds of these evidenced circumscribed or limited pneumatization. In one-third the pneumatization was normal. He examined as a control group persons who had no history of otitic infection. In 20 per cent of such normal persons he found changed pneumatization.

It would seem to me that while these studies are of interest the results reported remain questionable until an answer is found to the observation of Voss, for if there is subsequent replacement of cellular structure in a mastoid process following antrotomy, what conclusion can scientifically be drawn from the examination of skulls of patients who in early life suffered an otitic lesion? The possibility that healing without operation might also permit cellular development must be kept in mind.

Wittmaack [5] reiterates his point of view that during later life otitic lesions take form and specific characteristics from what happens to the temporal bone at birth. He calls attention to the three predominating types of bone tissue on which the otitic lesions take form. These are (1) the hyperplastic, (2) the purely exudative and (3) the combined types. There is one very pertinent comment in his report. He calls attention to a factor of which many who report operative findings as pathologic findings should take heed. He decries the diagnosis of pathologic changes in the bone made at operation. He calls attention to the fact that the very procedures of operation disturb the finer tissue relationships and create artefacts. He stresses that only from patho-

4. Loebell, H.: Ohroperation im frühen Kindesalter und Pneumatisations-Entwicklung, Ztschr. f. Laryng., Rhin. **21**:326, 1931.

5. Wittmaack, K.: Zur Frage der Bedeutung der Mittelohrentzündungen des frühesten Kindesalter für später, Arch. f. Ohren.-, Nasen- u. Kehlkopfh. **129**: 207, 1931.

logic studies of the temporal bone made with serial sections can a real estimate of the pathologic changes be made, and he frankly states that the roentgen examination can give better information than the study of bone spicules taken from the operative field.

I fully agree with these standards, yet should not forego the information won from the study of operative material, provided that in scientific reports it be clearly stated that the observations made are thus arrived at. In this country, otologic autopsy material, completely studied, is still too rare for one to forego the information available at the operating table. Final conclusions, however, had best be reserved for material handled according to Wittmaack's standards.

Husik [6] connects the type of bone presented with tendencies toward intracranial involvements. He accepts Wittmaack's divisions of the bone tissue. This is now beginning to be understood. A compact bone cannot coalesce; hence acute coalescent mastoiditis is impossible when sclerosed bone is present. On the other hand, sclerosed bone is often the underlying structure which, when an infection of the acute middle ear supervenes, most often gives an intracranial lesion as a sequela.

I shall again refer to the question of pneumatization when I discuss lesions of the petrosal pyramid.

The relationship of systemic and general diseases to otitic infections has long engaged otologic attention. On the one hand the otitic lesion is the irritator or starting point of a general systemic disease, like sepsis; on the other hand, it may cause the involvement of some visceral organ, like the lungs or the kidneys. Then there are the general systemic disorders on which is superimposed an otitic infection. As an example of the latter, the otitis occurring in diabetes may be cited.

Kinney [7] discusses the relationship of otitis to nephritis and differentiates two types of cases: 1. Those of focal or embolic origin, which affect only a few renal glomeruli. The patients present a hematuria as the only significant symptom, besides those arising from the otitis. They eventually recover, but often the hematuria lasts for months. 2. Cases in which there is an acute hemorrhagic glomerulonephritis. There are many glomeruli involved. The symptoms presented comprise hematuria, albuminuria and high blood pressure, and the patients have severe headache, edema and in some case even uremia. When these patients survive the acute onset during the first few days, prognosis as to life is good, provided the primary focus in the middle ear is eradicated.

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6. Husik, David N.: Sclerotic Mastoiditis and Intracranial Complications, Laryngoscope **42**: 519, 1932.

7. Kinney, C. E.: Laryngoscope **42**:356, 1932.

Similar observations were made by Okonogi,[8] whose studies comprise 30 cases of acute nephritis occurring among 357 cases of acute suppurative otitis. He traced a relationship between the intensity of the otitic infection and the renal lesion. He found that when the otorrhea was marked there was an increase in urinary abnormalities. Finally, when treatment favorably influenced the otitis, there was a favorable nephritic reaction as a result. He noted a rapid improvement in the condition of the kidneys after mastoidectomy.

With these views I coincide. I do not believe in delay in operative procedures when renal conditions supervene; and I too have noted the rapid improvement in the kidneys after a mastoidectomy has eradicated the purulent elements and their toxins from the body.

Special Clinical Types of Acute Otitic Infections.—There are a number of special types of otitic infection which deserve special attention. On the one hand, constitutional systemic diseases play a rôle and not only influence the course of the aural lesion, but materially affect the therapy required to effect cures. On the other hand, the aural lesion is of itself significant because it is the local manifestation of a general systemic disease. Finally, there are the systemic infections superimposed on otitic infection. I present the following, briefly considered:

Otitis occurring in children having diphtheria does not necessarily have to be a specific otitis. This has long been known. Bloch[9] again calls attention to this in reporting 3 cases of diphtheritic otitic infection with the specific organism as the causal agent. Chatellier[10] found the specific aural lesion in only a few, and of 2,378 cases of diphtheria observed from 1928 to 1932, only 2 required antrotomy. With these views LeMee[10] agrees, except that he found a larger number of cases of diphtheritic otitis among children with nasal diphtheria.

Mastoiditis in diabetes is always a problem. Voss[11] made an experiment which is of moment in this connection. He depancreatized dogs and then induced a purulent otitis media. He reported a more rapid development of the aural lesion in these animals than in controls likewise infected. From this it follows that early recognition and prompt eradication of the aural lesion are a necessity in diabetes. The results are good when surgical relief is prompt and is accompanied by

8. Okonogi, Shuzo, and Shunji, Susuki: Relation between Acute Otitis and Nephritis, Otologia **5**:295, 1932.

9. Bloch, André: Les otites de la diphtérie, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **18**:574 (Dec. 10) 1932.

10. In discussion of Bloch.[9]

11. Voss, O.: Klinische und histologische interessante Befunde diabetischer Ohrenkrankungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **29**:230 and 257, 1931.

systemic therapy to hold the sugar content of the blood within reasonable bounds (Sagols,[12] MacKenzie [13]).

Among the special clinical types of acute otitic infections none play a more important rôle than those caused by Streptococcus mucosus-capsulatus (pneumococcus type III). Sekulić [14] found that the symptom of greatest importance in mucosus otitis is the presence of an edema of the membrana tympani, which does not necessarily show marked redness. Paracentesis is followed by purulency, accompanied by a severe loss in hearing. When mastoiditis ensues, as it often does, there is rapidly spreading destruction of bone, which proceeds without much pain and often without a rise in temperature.

In this thumb-nail sketch of the clinical picture may perhaps be found the elements of a rational theory on which to base therapy. There are still many to whom the only indication for mastoidectomy in cases of otitis media caused by Streptococcus mucosus is the identification of this organism. These persons recommend operation irrespective of the clinical state of the patient, whether or not bony destruction is shown roentgenologically and whether or not the patient's tissue reactions, as denoted by studies of the blood, give evidence that the patients are resisting the infection.

True, many mucosal infections lead to meningitis. But with the newer methods of diagnosis—with the ability of roentgenologists to report the presence or absence of osteoporosis—reliance need not be placed entirely on the patient's subjective symptoms, and there is little cause for holding to a laboratory finding as an indication for operation.

Other facts, too, must be stressed. Many cases of mucosus otitis have cleared up without mastoidectomy. Operation, in some instances, has tended to spread the infection endocranially, especially when undertaken too early in the course of the disease, and quite a number of patients have been operated on unnecessarily. Lastly, there is the long record of many cases in which operation was performed and nevertheless meningeal infections developed.

In doubtful cases, when the purulency of the middle ear is in question and bacterial flora is desired for examination, Reiniger [15] punctures the membrana tympani with a long sterile needle, according to a method recommended by Neumann. I have disapproved of this type of

12. Sagols, H.: Mastoïdite et diabète, Ann. d'otolaryng., 1932, p. 575.

13. MacKenzie, George W.: Mastoiditis in the Presence of Unsuspected Diabetes; Report of a Case, Laryngoscope **41**:638, 1931.

14. Sekulić, Bözidar V.: Die Ohrenkrankheiten durch Pneumococcus mucosus, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **18**:789 (July 25) 1932.

15. Reiniger, A.: Ueber die diagnostische Bedeutung der Mittelohrpunction, Monatschr. f. Ohrenh. **66**:694, 1932.

procedure heretofore, and no convincing evidence has since been presented to change my point of view.

Kadiura [16] reports on 40 cases of mucosus otitis, with special reference to the biologic reactions of the bacteria. He discovered one atypical hemolytic growth, which sometimes was capsule-free, alongside of typical Schottmüller types. Kadiura believes that he has established transitional forms, both clinically and bacteriologically. That there are transitional types has been suspected, because occasionally there is found a Streptococcus mucosus-capsulatus of otogenic origin which culturally, especially in subcultures, takes on the metabolic characteristics of Streptococcus haemolyticus, while morphologically showing a capsule. These really are more streptococcus than mucosus forms, if I may be permitted the use of these descriptive terms.

Influenzal Otitis.—There have always been observed, during epidemics of influenza, specifically characteristic cases of an otitis which seems to constitute an entity and which is designated in the literature as influenzal otitis. Such cases are occasionally observed sporadically. The pathology of this condition has had various designations. I observed such cases in the epidemic which occurred several years ago, and described them in detail; other observers did likewise, but no one connected the observation with the prevalent influenza. To present studies from the recent epidemic abroad is interesting, and because of the general lack of appreciation of its distinctive character the report of Tanturri [17] becomes of moment.

The clinical diagnosis rests on the presence of a strongly expressed hyperemia of the membrana tympani. There is only a slight degree of sinking of the posterior superior meatal wall. Clinically, the patient presents marked pain and general symptoms of sepsis, often accompanied by chills. On paracentesis, the resulting discharge is scanty, and there is little relief of symptoms.

Following mastoidectomy the symptoms rapidly disappear, often within twenty-four hours, and then healing ensues. At operation Tanturri found no frank pus, but very hemorrhagic cells with an occasional necrotic spot. He concludes that early surgical intervention is a step in preventive treatment.

An identical report is made by Rejtö,[18] from Budapest, on the otitic influenza there. He reports cases of high fever, sepsis, etc., which at operation showed hemorrhagic bone with but little frank pus. In addi-

16. Kadiura, Kishirs: Klinische und bacteriologische Studien über Mucosus-Otitis, Otologia **5**:1001, 1932.

17. Tanturri, D.: Antro-mastoidite congestiva grippale, Rinasc. med. **9**:289, 1932.

18. Rejtö: Ueber die Eigenheiten der diesjährigan Influenza-Otitis, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **19**:73 (Aug. 25) 1932.

tion, Rejtö reports that he often found the lateral sinus wall diseased, but no demonstrable thrombus within it. He too recommends early operation and stresses the fact that the classic signs of mastoiditis usually looked for are not to be found. Rutenburg,[19] reporting on the epidemic of grip in Russia in 1927 and 1928, arrives at similar conclusions. During the mild epidemic of influenza in 1932 and 1933, I saw 2 cases which might fit into the classification set forth.

An unusual case of influenzal otitis is described by Otto Mayer.[20] He describes the sections made from a temporal bone taken from a patient with myringitis vesicularis. It was seen that there was no disease in the bone itself; that is, there was no true bone lesion present. Myringitis vesicularis is a hematogenous infection (as are all the influenzal lesions) which seems to develop in the area supplied by the deep auricular artery. Usually these lesions heal spontaneously. When recognized for what they are, and not mistaken for blebs on a bulging drum, no paracentesis should be done. If the lesion is a very virulent one, because of the anastomotic branches of the blood vessels the lesion in the blood vessel spreads on the inner surface of the tegmen tympani and sets up a meningitis as it did in Mayer's case.

Zygomatic Otitides.—Weill [21] has described a sharply circumscribed form of otitic infection, with the focus of a point of intense pain at or near the root of the zygomatic process. The temporal muscle bulges outward, and there develops over this area an edema which extends rapidly to the eye and involves the eyelids on the affected side.

The differential diagnosis consists in distinguishing these lesions from those of otitis externa with abscess formation on the anterior wall of the external auditory canal and from periauricular adenitis and arthritis of the temporomaxillary joint. Weill reports 2 cases.

Nevolnikova,[22] during the period between 1920 and 1928, studied 24 cases. He differentiates true otitic zygomatic infection from another form which he calls "the false." The diagnosis rests on the fact that the zygoma is involved at the same time that the mastoid process is invaded, although occasionally the mastoid involvement antedates the development of the zygomatic root. He states that on rare occasions the zygomatic involvement has been observed to occur independently of any mastoidal infection. The false or pseudo types of this lesion

19. Rutenburg, D.: Acute Otitis Media During Grip Epidemic of 1927-1928, Russk. otolaring. **24**:458, 1931.

20. Mayer, Otto: L'anatomie pathologique de la myringite vesiculeuse, Rev. de laryng. **53**:825, 1932.

21. Weill, G. A.: Le phlegmon zygomatique sous-temporal, Oto-rhino-laryng. internat. **15**:482, 1931.

22. Nevolnikova, L.: Zur Klinik der Zygomatitiden otogenen Ursprungs, Zhur. Ushn., Nos. i Gorl. Bol. **8**:527, 1931.

are simple superficial subperiosteal abscesses located in the area of the zygoma without involvement of this process. These sometimes result from the breaking through of an epidural abscess. Nevertheless, whatever type of lesion is present, surgical intervention is the means to procure healing.

Yates [23] reports on a subacute form of acute otitic infection in which there is no rupture of the membrana tympani, but the pus is discharged through the eustachian tube into the pharynx. The usual swelling and edema which occlude the eustachian tube are absent because of the lack of intensity in the tissue reactions to the infection, and the pus finds a ready egress through the preformed channel of the tube. Such cases are often overlooked.

In this connection the observations of Engel [24] are significant. He states that no cases have been reported in which the route of the infection has been definitely established at autopsy. In a case cited he found a purulent tract from the ear emerging in the peritonsillar area. On the insertion of a probe into the tract through the peritonsillar opening, after removal of the tonsil, he introduced a eustachian bougie through a catheter into the eustachian tube, injected a radiopaque substance (iodized sesame oil 40 per cent) and then examined the patient roentgenographically. Thus he was able to trace the tract of purulency from the ear to the pharynx. Engel [25] reports a similar case except that this showed a gravity abscess in connection with mastoiditis. On the injection of iodized oil and with a probe in position, roentgen examination demonstrated that the peritonsillar collection of pus developed from the mastoiditis.

Erysipelas.—When erysipelas appears after mastoidectomy it is usually believed to be a complicating infection of the operative area. Segall [26] contends that there is a clinical entity—otitis media erysipelatosa. In this lesion there need be no exudate in the middle ear, nor need a perforation take place in the drum head.

Grossman,[27] citing his own observations, takes issue with this description. He too concedes the possibility that this is a clinical entity and calls attention to the classic description of it in Denker and Kahler's

23. Yates, A. Lowndes: Subacute Otitis Media, Brit. M. J. **2**:647, 1931.

24. Engel, F.: Peritonsillarer Abscess Otogenen Ursprungs, Zentralbl f. Hals-, Nasen- u. Ohrenh. **19**:303 (Oct. 10) 1932.

25. Engel, F.: Otogener Senkungsabszess im Rachen u. röntgenologische Aufdeckung der Wegleitung, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **30**: 322, 1932.

26. Segall, Walter: Untersuchungen über die Ausbreitung des Schleimhautcrysipels vom Nasen-Rachenraum auf die aüssere Haut über das Mittelohr, Wien. med. Wchnschr. **81**:1592 and 1684, 1931.

27. Grossman: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **17**:575, 1931.

textbook. Grossman contends that it takes three or four days for the inflammation which starts in the pharynx to spread outward along the lymph spaces of the latter and finally appear externally at the surface over the ear. Segall's case showed an eruption in one day. The point made by these authorities is that pain in the throat is the first expression of the disease. To the controversy Eisinger [28] adds that during the early stage there is often swelling of the lymph glands of the neck, which he holds of diagnostic importance.

Infantile Otitis.—Lierle and Potter,[29] Voss,[30] Cone,[31] Koenigsberger [32] and Druss [33] have presented interesting reports on infantile otitis and its relationship to gastro-intestinal disturbances. There seems to be a clearer comprehension of this subject than formerly. Without detailing minor differences, the present consensus is that a relationship exists between the two conditions, the one in the ear and the disturbed gastro-intestinal function. Today no one of authoritative rank advocates operation on the ear unless a diagnosis of otitis has first been established.

Timing the Operation in Acute Cases.—The time at which to intervene in acute mastoiditis has often been discussed. Many hold the view, and I count myself among these, that better results are obtained from operation if one waits for a "walling off" process to take place. There is then less likelihood of a spread of the infection endocranially.

Coulet [34] makes a report on fatalities in relation to the time of operation. Among patients operated on in the first week of the mastoiditis, he had no fatalities. There was 5 per cent mortality among those operated on in the second week, 6.5 per cent among those who underwent mastoidectomy in the third week, and 13.7 per cent among those who came to operation during the fourth week and later. His material comprises 100 cases.

Coulet's indication for operation rests on diagnostic data comprising deep-seated spontaneous earache, which is throbbing and which does

28. Eisinger, Karl: Ueber Veränderungen des Lumbalpunktates bei Erysipel, Wien. med Wchnschr. **81**:1564, 1931.

29. Lierle, D. M., and Potter, J. J.: Factors Influencing the Course of Mastoiditis in Infancy, Ann. Otol., Rhin. & Laryng. **40**:788, 1931.

30. Voss, O.: Statistiches und Klinisches zur Otitis media im Kindesalter, Ztschr. f. Laryng., Rhin., Otol. **21**:313, 1931.

31. Cone, A. J.: Incidence of Acute Otitis Media in Infants and in Young Children During 1930-1931, Arch. Otolaryng. **10**:797 (Dec.) 1931.

32. Koenigsberger, E., and Trepel, G.: The Clinic of Obscure Mastoid Infections in Infants, Klin. Wchnschr. **11**:1392 and 1433, 1932.

33. Druss, Joseph G.: Infection of the Ear in Cases of Infection of the Gastro-Intestinal Tract in Infants, Am. J. Dis. Child. **43**:356 (Feb.) 1932.

34. Coulet, Gaston: Remarques sur les indications opératoires de mastoïdites aiguës, Rev. de laryng. **52**:616, 1931.

not abate after paracentesis, accompanied by unilateral (homolateral) headache, continuous fever and a purulent otorrhea. The presence of trigeminal pain or paralysis of the face or other regions is an indication for immediate operation.

On the other hand, Crow [35] opposes operation early in the disease and stresses the fact that at that time it is usually unnecessary and only rarely justified by threatening symptoms.

Conclusions cannot be drawn from reports which do not give full details of the symptoms, roentgen examination, blood picture and general physical examination. Certainly to base indications for operation on "time," in spite of the mortality figures given, would lead to many unnecessary surgical procedures, nor has any evidence yet been produced that a period of waiting for both coalescence to complete itself and nature to wall off the purulency tends to jeopardize the welfare of the patient.

Technic in Acute Cases.—There are some newer suggestions as to technic that are noteworthy. Shambaugh [36] wants to confine his operative procedure to the removal of diseased bone only. Consequently, the antrum is occasionally not opened. At other times the operation is confined to the tip or the lower part of the mastoid process. Shambaugh may have had good enough results from this procedure to warrant so difficult an individualism of his surgical technic. How to determine with the naked eye where diseased bone ends and normal bone commences would seem to be a stumbling block to the general adoption of this view, and the objective of postauricular drainage is certainly not attained when the antrum remains unopened. It has long been known that the simple opening of the cortex will stop the mechanics which play a rôle in producing a coalescence, but it has not been accepted that such a procedure—that is, the stoppage of coalescence—is the objective aimed at in simple mastoidectomy.

In Italy there has developed a school of men who perform a mastoidectomy, cut a flap into the meatal canal wall after reducing the bony meatal wall down almost to the drum head, then sew up the postauricular wound in the skin and watch the granulation and healing of the mastoidectomy through the external orifice of the auditory canal. Torrini [37] discusses the advantages and disadvantages of this method of procedure and reports on 200 cases in which he operated. He claims

35. Crow, Douglas A.: A Note on Premature Mastoid Drainage, Brit. M. J. **2**:1104, 1932.

36. Shambaugh, George E.: Acute Otitis Media Complicated by Mastoiditis, S. Clin. North America **12**:137, 1932.

37. Torrini, U. L.: Vantaggi e svantaggi della chiusura immediata della ferita retro-auricolare nelle mastoiditi acute, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **20**: 145 (March 25) 1933.

better cosmetic results and shorter after-treatment, but admits that the usual open method of operation permits better postoperative control of the wound. At the congress where the cases were reported, Caliceti [38] contended that this method of procedure prevented the formation of postauricular fistula. Attore [39] reported on the first 100 cases in which he used this method. His results were as follows: Eighty-eight patients were cured, 12 were still ambulatory at the time of his report and 3 had required reoperation. Except that this technic had been advocated for chronic mastoiditis, it is quite similar to that advocated by the English surgeon, Heath; in these cases, however, there is no epitympanic lavage. Bárány devised a similar procedure and demonstrated it while visiting America several years ago.

CHRONIC LESIONS

Cholesteatoma.—It has been taught that primary cholesteatoma (true type) is congenital. Yet Massione [40] believes that primary cholesteatoma can arise from a chronic otitis, due to a degenerative process following from extravasations of blood and new growth of connective tissue. This eventually undergoes degenerative processes and a cholesteatoma thus develops. His views are further at variance with accepted pathologic teaching in that he does not believe a radical mastoidectomy necessary if the cholesteatoma can be recognized.

Again, Lange [41] presents observations at variance with accepted theory. He does not think that the conception that epidermis grows into the middle ear spaces through a perforation can be maintained. He points to the fact that even a short time after the commencement of infection the epidermis grows over the short process of the ossicle and toward the outside into inflammatory thickened mucous membrane; even the ligamentum mallei anterius shows epithelial growth medially. Thus an encysted cholesteatoma can form. He points to the possibility of its occurrence even on pneumatized bone. Though I do not dispute or question his observation, I believe that he is dealing with pseudocholesteatoma and not with real cholesteatoma.

The diagnosis of cholesteatoma by laboratory methods is still an interesting open question. Rejtö [42] makes a further report on the chemical diagnosis of this lesion and adds observations on 28 additional

38. Caliceti, in discussion of Torrini.[37]

39. Attore, Guglielmo: The First Hundred Cases Operated on According to the Method of Torrini, Arch. ital. di otol. **43**:398, 1932.

40. Massione, Giuseppe: Ulteriore contributo all'istogenesi del colesteatoma dell'orecchio, Atti d. clin. oto-rino-laringoiat. d. r. **29**:315, 1931.

41. Lange, W.: Tief eingezogene Membrana flaccida und Cholesteatoma, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:575, 1932.

42. Rejtö, Alexander: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **19**:220 (Sept. 25) 1932.

cases. He finds that a rise in the cholesterol content of the blood is not produced by the cholesteatoma, but is the expression in the blood of a destruction of bone. He postulates that when there is a rise in the cholesterol content of the pus from the ear, with no related rise in that of the blood, it is possible to prognosticate little destruction of bone. This would explain the finding of Dean,[43] who often observed a raised cholesterol value when no cholesteatoma was present. In his cases the bony lesions conceivably might account for this finding.

A contrary opinion is also expressed by Peroni,[44] who examined 15 patients, ranging in age from 6 to 40 years, all of whom gave demonstrable evidence of cholesteatoma yet showed no hypercholesteremia. These results are controverted by Bernowitz,[45] whose findings led him to hold an opposite view.

Whatever the final determination as to the value of the chemical examination of cholesteatoma will be, there is a more definite means at hand to furnish information as to the presence or absence of this lesion. This means is furnished by roentgen examination. Volkmar [46] examined 35 patients and compared his roentgen findings with the observations made at operation. In 23 cases the diagnosis based on roentgen examination was correct. In 7 of the remaining 12 cases, the cholesteatomas were so small and the destruction of bone so limited that the films did not detail them. There were, however, 5 well developed cases which failed to be diagnosed roentgenologically.

Clinical Observations in Chronic Cases.—Schwarz [47] gives an interesting clinical study of Shrapnell's membrane. He believes that he can differentiate between a retraction of this membrane which is temporary and one which cannot be restored to normal by using the Siegel otoscope or through inflation. When the retraction of Shrapnell's membrane is fixed, it is an irreparable condition. The condition is found in children as well as in adults. It is usually bilateral. A significant observation is that in about 88 per cent of cases the change in Shrapnell's membrane was accompanied by the finding of a nonpneumatized mastoid process. Schwarz observes that in all cases of change in Shrapnell's membrane there is a loss of resistance to infection in the mucous membrane of the middle ear; the retraction of this membrane generally speaks for the evolution of a chronic infectious process of the middle ear.

43. Dean, L. W.: Cytological and Bacteriological Studies of Otitic Diseases, Laryngoscope **42**:857, 1932.

44. Peroni, A.: Cholesteatoma and Cholesterinaemia, Arch. ital. di otol. **43**: 72, 1932.

45. Bernowitz: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **17**:847, 1931.

46. Volkmar, Herbert: Rontgenerfahrungen bei Cholesteatomen des Mittelohres, Ztschr. f. Laryng., Rhin., Otol. **21**:424, 1931.

47. Schwarz, M.: The Retraction of Shrapnell's Membrane and Its Clinical Significance, Arch. Ohren-, Nasen- u. Kehlkopfh. **131**:16, 1932.

I am inclined to disagree with this reasoning. It may be accepted that permanent changes in Shrapnell's membrane, the formation of adhesions and the consequent changed blood supply may be a factor which causes an infection to locate itself in the area of the membrana flaccida. The finding that under the conditions described by Schwarz there is accompanying sclerosis of bone (88 per cent of his series) is more probably the reason why chronicity develops. Sclerosed bone cannot be the seat of an acute coalescing process. Once infection has taken place, and nonpneumatized bone structure is present, there can evolve only a chronic infection of the middle ear.

Jobson [48] believes that the presence of a cholesteatoma tends toward the formation of sequestra. This view is not in accord with classic conceptions. The cholesteatomatous mass is surrounded by rarefied strata of bone which, in its turn, has eburnized or sclerosed bone about it. Evidence as to the mechanics by which sequestration could take place is lacking.

Therapy.—There are the usual number of articles on therapy. They present, in the main, nothing new. The recommendation of vaccine therapy is limited to selected cases in which the mucous membrane is the seat of a chronic infectious process. The use of strong solutions of alcohol where cholesteatoma is suspected is recommended.

Mossböck [49] uses and recommends the use of chlorine gas in uncomplicated cases of chronic infections of the middle ear. He uses equal parts of chlorine gas and air and introduces this mixture into the external auditory canal after the middle ear has been carefully cleansed. The gas is kept in place by a glass olivary stopper at the external auditory orifice for five minutes. He has observed no bad effects and reports some good results. During the treatment a gauze pad soaked in a 10 per cent solution of sodium hydroxide is put in the mouth as a precaution and a protective measure.

Zinc ionization is by far the most popular measure reported during the last year. I shall detail some of the findings reported. Lacaille [50] used it in 9 cases with good results in only 3. On the other hand, Asherson [51] observed a cure in 240 cases, or 95 per cent of those in which he used it. He thoroughly cleanses the ear first. He selects only cases of central perforations, with no polyps, and there must be no cho-

48. Jobson, T. B.: Cholesteatoma in Aural Suppuration: Its Relation to Prognosis, J. Laryng. & Otol. **47**:380, 1932.

49. Mossböck, F.: Die Behandlung chronischer Schleimhauteiterungen des Mittelohres mit Chlorgas, Wien. klin. Wchnschr. **46**:76, 1933.

50. Lacaille, A.: Valeur de l'ionization au zinc dans le traitement des suppurations des cavités d'évidement, Ann. d'oto-laryng., 1931, p. 667.

51. Asherson, M.: Zinc Ionization in the Treatment of Suppurative Otitis Media, Laryngoscope **42**: 141, 1932.

lesteatoma present. Lierle and Sage [52] used the zinc ionization method after a radical mastoidectomy which failed to result in a healed ear.

These authors made experimental studies of the zinc ionization process to determine how it produces its effects. They contend that there is a remote possibility of a deposit of zinc in living tissue. The question cannot be considered as settled because the observers based their reports on spectroscopic findings rather than studies of tissues. They report that the electric current used has little if any antibacterial value. They imply that the good results obtained are the result of a deposit of zinc sulphate in the tissues.

Walker,[53] who reports generally favorable results, advises that if good results are not obtained by the second treatment, there is no use in persisting with this method. He applies the treatment to ears which have a large perforation, an open eustachian tube and no necrosis of bone. He reports cure in about 50 per cent of cases.

Reynolds [54] restricts the use of this method of therapy to early cases. Among 36 cases, he obtained cure in 55 per cent and improvement in 42 per cent. In 3 per cent there were no results.

Asherson [55] uses a combination of 99 per cent boric acid and 1 per cent iodine powder, which he instils after thorough cleansing with alcohol. He reports good results from the use of this method in about 300 cases.

Epitympanic Operation.—There are an unusually large number of reports on surgical therapy. The conception has gained ground that eburnized bone (lack of pneumatization) is not a pathologic lesion, but the after-effect of an incident at birth (Wittmaack) and that, therefore, in performing a radical mastoidectomy it is unnecessary to remove all this compact bone, which contains no diseased tissue and does not constitute a threat to life. The conception is gaining ground that operation should be limited to the removal of pathologic tissue. Hence there are more reports of epitympanic operations than before. The epitympanic operation was popularized in America by Tobey, who reaches his objective through the external auditory canal.

Babbitt [56] also uses the epitympanic procedure, working either from a posteriorly located perforation or from an incision which he places in

52. Lierle, D. M., and Sage, R. A.: Underlying Factors in the Zinc Ionization Treatment of Middle Ear Infections, Ann. Otol., Rhin. & Laryng. **41**:359, 1932.

53. Walker, I. D.: Value of Zinc Ionization in Selected Cases of Suppurative Otitis Media, Arch. Phys. Therapy **13**:90, 1932.

54. Reynolds, G. S., and Whitney, E. L.: Summary of One Hundred Cases of Otitis Media Treated by Zinc Ionization, Arch. Phys. Therapy **13**:527, 1932.

55. Asherson, M.: Iodine Powder in the Treatment of Chronic Otorrhea, Lancet **2**:630, 1931.

56. Babbitt, J. A.: A Modified Attic Drainage in Chronic Suppurative Otitis Media, Ann. Otol., Rhin. & Laryng. **40**:348, 1931.

the posterior part of the membrana tympani. Through this he locates the antrum, removes granulations and obtains a cure in about 70 per cent of cases. His patients range in age from 3 to 75 years. The hearing is good after operation.

Komuro [57] also uses this limited operative procedure. He believes that the cases should be carefully selected. This operation shortens the period of treatment, and there is less loss of hearing. He places the indications for the operation as follows: (1) in relative light grade infection of the attic and antrum when there is good hearing; (2) when labyrinthine symptoms supervene in such a case; (3) when there is great loss of hearing on the opposite side; (4) when a radical operation is being performed and the pathologic changes are limited to the epitympanic cavity and antrum; (5) when there are headache and a pulsating light reflex without an acute exacerbation, and (6) when the operation may be of value as a preventive measure. He recommends entrance either through a postauricular incision or, like Babbitt, through the external auditory canal.

Brunetti [58] discusses the epitympanic operation. Many surgeons have devised a technic by which one enters the epitympanic space through the external auditory canal without opening the mastoid cortex. Brunetti comments on a similar technic advocated by Torrini who, however, reaches the epitympanic space from the cortex. Brunetti, in criticism, stresses that neither method of technic is applicable to an acute condition. On the contrary, to incise the external auditory canal in an acute condition is not only harmful but useless. The disadvantages of the operation are stenosis of the canal, chondritis and perichondritis. Brunetti recalls Voss' advice not to cut flaps in the epitympanic operation.

Citelli [59] stresses the importance of after-care as most important in radical operations on the mastoid. Immediately after operation he inserts tampons. He takes these out a few days later and thoroughly dries the ear with cotton pledgets soaked in concentrated solution of alcohol, after which he sprays boric acid over the area. He places a drain in the aditus and another in the external auditory canal. After fifteen days there is cicatrization, and after four weeks healing is complete.

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57. Komuro, Kaname: Die konservative Radikaloperation der chronischen Mittelohreiterung. Otolagia **3**:391, 1930; abstr., Zentralbl. f. Hals-, Nasen- u. Ohrenh. **18**:40, 1932.

58. Brunetti, Frederico: Ancora sull'antrotomie epitimpanale. Arch. ital. di otol. **43**:692, 1932.

59. Citelli, S.: Su un nuovo metodo assai utile e simplice di medicazione della radicale dell'orrecchio medio. Oto-rino-laring. ital. **2**:435, 1932.

Jessen [60] compares the subjective and the objective changes before and after the radical operation. His observations cover 205 radical operations. He distinguishes between operations performed in the "cold" state, when no acute, local or systemic signs are present, and those performed in the "hot" state, when an acute clinical picture is superimposed on the chronicity in the middle ear spaces. Seventy-five patients were operated on in the "cold" state. In 130 the condition was of the acute type. In the second class were many children. The results of operation in both types of cases were about the same. A comparison of subjective symptoms before and after operation shows that good results predominate after operation. Fifty per cent of the patients failed to show a dry ear, one-fourth of them having a more severe discharge than before operation. Anatomic conditions were better in 41 per cent; in 10 per cent they were worse. Of 71 patients operated on according to the Bárány method 58 per cent showed good results, 3 per cent poorer results and the remainder only partial improvement. There were 84 cases of cholesteatoma. Of these 35 per cent gave good results. In 10 per cent the results were poor. For the last four years Jessen has operated according to the method of Bárány (an epitympanic operation through the canal). The following results were obtained in 71 cases: In 45 the external canal was maintained. There were 8 cases of recurrence, of which 2 showed cholesteatoma, not identified at the first operation, and 5 revealed no cholesteatoma either before or after the operation. Complete epidermization was obtained in 45 per cent of the cases. A complete lack of epidermization was noted in 36 per cent. Hearing was poorer in 2 instances.

Pressman [61] studied the hearing after operation in 372 cases, and concluded that the hearing is not worse after a radical operation than after continued conservative treatment. Fairly good hearing is no contraindication to a radical operation.

Late complications after a radical mastoidectomy have rarely been reported. Tamari [62] stresses this and reports 9 cases. He holds that the radical mastoidectomy is really a prophylactic measure against endocranial complications. Late complications are mostly acute exacerbations. They are due to (1) incomplete operative procedures, (2) atresia forming postoperatively, (3) tissue bridges and scarifications, which obstruct the free egress of detritus and discharge, and (4) reinfection, favored by the foregoing factors.

60. Jessen, J.: Les résultats de l'opération radicale de l'oreille moyenne, Acta oto-laryng. **16**:459, 1931.

61. Pressman, J. J.: Residual Hearing After Radical Mastoidectomy, Laryngoscope **41**:808, 1931.

62. Tamari, M.: Late Complications After Radical Operation, Monatschr. f. Ohrenh. **66**:286, 1932.

Haubrich [63] studied the effect of a radical mastoidectomy in children. He observed 83 cases among patients ranging in age from 1 to 15 years. In spite of the general opinion to the contrary, he holds that in carefully selected cases the radical operation may be performed in childhood. His results were good in most cases. In 7.5 per cent there were endocranial complications.

On the other hand, Yearsley [64] calls attention to the psychic effect of radical mastoidectomy in children. He observed deleterious influences on the children's intellectual development, owing partly to poor hearing. The children became nervous, irritable and inattentive and showed a lack of concentration. Yearsley therefore advises the avoidance of a radical operation and stresses the local treatment of the chronic disease of the middle ear.

When one considers that the almost inevitable consequence of an unhealed chronic infection of the middle ear is a serious complication, and as the finding of sclerosed bone is a factor in chronicity on the one hand and in intracranial complications on the other, it would seem that in children who have been carefully and thoroughly studied and treated without favorable results an epitympanic radical operation should be performed. From the standpoint of intracranial complications such a procedure has valuable prophylactic elements.

SINUS THROMBOSIS

There is practically nothing new in the literature on sepsis and sinus thrombosis. Cases which varied clinically and in which healing followed eradication of the focal lesion in the sinus are reported, as well as failures which came to autopsy. A complete study involving 50 patients observed during the period from 1907 to 1931, is reported by Fraser.[65] Of these patients 30 (60 per cent) recovered and the remainder died. Twenty-two came under observation with an acute lesion, and of these 15 recovered and 7 died. Three died of meningitis and 1 of cavernous sinus thrombosis; 1 died as a result of metastatic lesions and jugular thrombosis. In 28 there were associated chronic tympanic lesions. Of these patients 15 recovered. The associated lesions were as follows: meningeal involvement in 2, cerebellar abscess in 2, labyrinthitis in 3 and jugular thrombosis in 1.

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63. Haubrich, Karl: Erfahrungen der Marburger Universitäts-Ohrenklinik über die vollständige Aufmeisselung der Mittelohrräuma bei Kindern, Dissert., Marburg, 1932, p. 43.

64. Yearsley, Macleod: The Mental Effect of the Complete Mastoid Operation in Children, Lancet **2**: 613, 1932.

65. Fraser, J.: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **19**:298 (Oct. 4) 1932.

Boies,[66] in his series of cases, separates those of thrombosis before mastoidectomy from those in which the sinus thrombosis occurred postoperatively. The mortality was 25 per cent. Potts[67] reports 63 cases, with 9 deaths. The Tobey-Ayers test gave no positive results. Lillie and Williams[68] call attention to the symptom of severe periodic lancinating pain, occurring mostly at night. In one case the pain started at the angle of the jaw and spread to the cheek, toward the zygoma and over the side of the head. These observers believe the pain due to pressure of glandular tissues on the ramus jugularis vagi. Portmann[69] calls attention to the finding that a thrombosed sinus pulsates and a normal one does not, an observation that I can substantiate.

Grossman[70] reports a case which presented a thickly indurated swelling of the neck, beginning over the lower half of the mastoid process and extending downward to the middle of the neck. On incision, pus was drained and the vessel ligated. The condition within the mastoid having been treated, the patient recovered. Layera[71] reports a case of marked sepsis in which no thrombus was demonstrated. Mygind[72] had 98 cases of sinus thrombosis among 1,000 cases of diseased mastoids. Of these, 55 per cent cleared up. Jugular ligature was undertaken in the earlier cases but not in the more recent one. Mygind reports no appreciably poor results in the latter group.

Fremel[73] reports 5, Schlander[74] 3 and Maybaum[75] 20 cases of primary thrombosis of the bulb. As Fremel states, the definition of primary thrombosis of the bulb is not definitely fixed, and many cases so termed would not be included in this group in a strict scientific

66. Boies, L. R.: Lateral Sinus Thrombosis; A Review of 184 Cases with Special Reference to the Post-Operative Type, Ann. Otol., Rhin. & Laryng. **41**: 227, 1932.

67. Potts, J. B.: Thrombosis of the Lateral Sinus, J. A. M. A. **98**:379 (Jan. 30) 1932.

68. Lillie, H. I., and Williams, H. L.: Pain as a Symptom of Thrombosis of the Internal Jugular Vein, S. Clin. North America **12**:883, 1932.

69. Portmann, Georges: Considérations sur le traitement de phlébites et thrombo-phlébites sinuso-jugulaires, Rev. de laryng. **52**:523, 1931.

70. Grossman, Fritz: A Case of Unusual Parietal Sinus Thrombosis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:164, 1931.

71. Layera, Juan, and Torres, Ignacio: Ein Fall von otogener Septicopyaemia ohne Thrombosis des Sinus laterales, Prensa med. argent. **18**:117, 1931.

72. Mygind, S. H.: Treatment of Otogenic Sepsis and Sinus Thrombosis, Acta oto-laryng. **16**:474, 1931.

73. Fremel, F.: Ueber primäre isolierte Bulbuserkrankungen, Monatschr. f. Ohrenh. **66**:1234, 1932.

74. Schlander, Emil: Irrigation of the Bulb, Wien. med. Wchnschr. **81**:1591 and 1683, 1931.

75. Maybaum, J. L., and Goldman, J. L.: Primary Jugular Bulb Thrombosis; A Study of Twenty Cases, Arch. Otolaryng. **17**:70 (Jan.) 1933.

classification. Perhaps that is why Grossman [76] questions 9 of Maybaum's cases.

Rott [77] asks why the jugular vein should be ligated in cases of lateral sinus thrombosis. He thinks that this is done more because of tradition than because of a rational theory. He contends that in treating an infection of the lateral sinus the first requisite is the removal of the focus of infection. He observes that ligature of the jugular vein does not prevent the absorption of toxins and bacteria into the general system. It is therefore unnecessary. He reserves ligation of the jugular vein for cases in which the jugular vein is the seat of the thrombus; then the vein should be resected. Mygind has a similar opinion, but Portmann holds the opposite point of view, and ligates all internal jugular veins.

The use of a fixation abscess to help combat sepsis in cases of sinus thrombosis is reported by Canuyt [78] and Abbate.[79] The latter reports its successful use in 10 cases.

As having a bearing on the measurements taken in the Tobey-Ayers test, the studies of Edwards [80] are important. He studied the anatomic variations of the cranial venous sinus in 50 skulls. Forty-six (92 per cent) showed a difference in the diameter of the two venous channels. In 25 the right side, and in 21 the left side, was the wider. The variation is greater when the wider channel is on the right than when it is on the left. The left venous channel is somewhat larger than the right. Since the venous outflow from the cranial cavity must go through both venae jugulares internae, the measurement of both sinuses must influence the results of the Tobey-Ayers test. With compression of the wider vein, the venous stasis and the fluid pressure are higher than with compression of the narrower sinus. This is the reason why measurements of the cerebrospinal fluid show higher pressure figures when pressure is exerted on the right side than when the left side is compressed.

After resection of the lateral sinus and ligature or resection of the internal jugular vein circulatory disturbance is unusual. It is important

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76. Grossman, Fritz: Zentralbl. f. Hals-, Nasen- u. Ohrenh. **20**:562, 1933.

77. Rott, O. M.: Why Ligate the Jugular Vein in Cases of Lateral Sinus Thrombosis? Arch. Otolaryng. **14**:272 (Sept.) 1931.

78. Canuyt, Georges: Fixation Abscess, J. Laryng. & Otol. **47**:235, 1932.

79. Abbate, R.: Gli ascessi di fissazione nelle setticemie otogene, Ann. di laring., otol. **31**:256, 1931.

80. Edwards, E. A.: Anatomic Variations of the Cranial Venous Sinuses; Their Relation to the Effect of Jugular Compression in Lumbar Manometric Tests, Arch. Neurol. & Psychiat. **26**:801 (Oct.) 1931.

to know that this finding is recorded. The observations of Ohnacker [81] are of moment. In a report of 4 cases and comment on another, he holds that cerebral symptoms following resection are due to a narrowed outflow channel on the healthy side. Occasionally pathologic changes in the sinus transversus are responsible. The patients present a heightened intracranial pressure and symptoms which simulate meningitis or abscess of the brain.

THE PETROSAL PYRAMID

This is my first survey to include a study of the current literature on the lesions of the petrosal pyramid. Before a list of the year's contributions, it is proper that an introductory note be given, sketching the topic.

For some years there have been reports of sporadic cases, unrecognized clinically for the most part, which eventually came to autopsy and were studied macroscopically and microscopically. The observers found and reported the lesions in the petrosal pyramid as interesting and unusual. These lesions were traced as purulent tracts leading from the middle ear to the meninges. Sometimes at operation a surgeon was led beyond the usually exposed areas of the mastoid cavity to follow purulent tracts which he searched with varying degrees of success. These clinicians, not understanding the relationship of what they found to the symptomatology which the patients presented, nor comprehending the sequence of events until the well known clinical picture of meningitis supervened, were not in a position to see the general indications for operative procedures on the petrosal pyramid.

The Gradenigo syndrome has engaged otologic attention for years. Its relationship to meningitis was understood, and when cases showing a Gradenigo syndrome came to autopsy many reports of purulent involvement of the petrosal pyramid were recorded. Perhaps that accounts for the conception which became general in 1930, that the syndrome was the outstanding characteristic symptom of suppuration of the petrosal pyramid. This has subsequently been proved untrue.

Meanwhile, during the transitional period in these conceptions, Profant [82] published his work. Closely following him, Eagleton [83]

81. Ohnacker, Paul: Zirculatorisch bedingte Hirnerscheinungen im Anschluss an Sinus- und Jugularisausschaltung, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **131**: 1, 1932.

82. Profant, H. J.: Gradenigo's Syndrome with a Consideration of "Petrositis," Arch. Otolaryng. **13**:347 (March) 1931.

83. Eagleton, W. P.: Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex, Tr. Am. Laryng., Rhin. & Otol. Soc., 1930, p. 338; also, with additional case reports in Arch. Otolaryng. **13**:386 (March) 1931.

reported his operation for unlocking the petrosal pyramid as a means of eradicating a lesion (osteomyelitis) in the pyramid and preventing the development of a meningitis.

Almour's and my own [84] first report then appeared in this country, at almost the same time that Voss [85] made public his studies. Voss, however, does not present the clinical picture of the petrosal lesion. In the report of Almour and Kopetzky the clinical picture is definitely established and the symptomatology analyzed. Significant signs consist of pain along the first branch of the fifth nerve (orbital, intra-orbital or retro-orbital pain), a low grade fever and a reappearance of the discharge from the middle ear after a period of cessation, or its continuance without a period of stoppage. The insignificant diagnostic value of the Gradenigo syndrome was particularly stressed. After a critical study of the lesion, Almour proposed a specific technic to meet the surgical requirements presented by the lesion. Then followed the contribution of Frenckner.[86] These articles gave the lesions of the pyramid their scientific status in otology.

To understand the lesions in the petrosal pyramid one must remember that they develop on a bony structure similar to that present in the mastoid process. Most investigations have shown that a well pneumatized mastoid process usually presents a pneumatized petrosal pyramid. Diploic and sclerosed bone and that of mixed type accompany similar findings in the mastoid process.

Pneumatized bone, under the same assault to which it is subjected in the mastoid process, coalesces with the development of a lesion designated as osteitis. The diploic bone, with its cancellous tissue, often containing bone marrow, succumbs to a lesion known as osteomyelitis.

From the four early reports it is evident that Eagleton is dealing with osteomyelitis. Almour and Kopetzky's reports are concerned with coalescent pneumatization (osteitis). Neither Voss nor Frenckner differentiates or classifies his cases on the basis of the underlying anatomic pathology. There is an ample bibliography on the anatomic structure and the cellular development of the petrosal pyramid in the work already cited.

An outline of the suggested surgical procedures is necessary. In a brief description, Eagleton advocates the removal of the posterior

84. Kopetzky, S. J., and Almour, R.: The Suppuration of the Petrous Pyramid; Pathology, Symptomatology and Surgical Treatment, Ann. Otol., Rhin. & Laryng. **39**:996, 1930; **40**:157, 396 and 992, 1931.

85. Voss, O.: Pyramidenspitzeneiterungen (Petrositis) an der Hand von 12 Selbstbeobachteten Fällen, Acta oto-laryng. **15**:469, 1931.

86. Frenckner, P.: Some Remarks on the Treatment of Apicitis (Petrositis) with or without Gradenigo's Syndrome, Acta oto-laryng. **17**:97, 1932.

buttress (the tegmen celluli, sinus plate, bone of the sinodural angle and bone over Trautmann's triangle) and of the anterior buttress (root of the zygoma, part of the zygoma, part of the squamosa and part of the anterior canal wall), the removal of the entire tegmen and the elevation of the dura of the middle cranial fossa until the petrous apex is brought into view. A curved sharp hook is now used to remove the bone and enter the pyramidal tip.

Voss attacks the lesion by extending the procedures of the radical mastoidectomy as far as possible to lay bare the epitympanic space, in the hope of reaching the lesion.

Frenckner's technic consists in entering the petrous apex through the arch of the superior semicircular canal. Widely exposing the epitympanic space, he employs a fine curet to bore his way into the depths, removing the cancellous tissue contained in the quadrant between the horizontal and the superior canals. He follows the tract that he makes underneath the arch of the superior canal, working inward until the tip is entered and the pus evacuated.

The technic which I advocate and have used, devised by Almour, comprises the performance of a radical mastoidectomy as a preparatory step, unless a fistulous tract is found in the epitympanic space, which is searched before the radical operative steps are undertaken. Following the radical mastoidectomy, the tensor tympani having been removed, the tip is entered through the roof of the tympanic orifice of the eustachian tube.

Since the reports referred to appeared, Lillie [87] has reported 2 cases in which he obviated the necessity of a radical mastodiectomy because he found leads into the purulent area of the tip in the epitympanic space.

Meanwhile, Ramadier, Guillon and Becker [88] suggested a technic which was never employed on the living subject but which they worked out on the cadaver. Through the usual mastoid incision, carried slightly under the auricle, a radical mastoidectomy is performed. The membranous canal is then completely removed from the bony canal, and the soft parts are freed so as to expose the glenoid fossa. The capsule of the mandibular joint is then separated from the anterior canal wall, and the entire bony canal wall and part of the floor are removed. The tegmen and tegmen antri are next removed, and the posterosuperior part of the glenoid tubercle is resected. These procedures bring the tympanic tubal orifice into view, and the anterior tympanic wall of the middle ear

87. Lillie, H. I.: Suppuration of the Petrous Pyramid; Report of Two Cases, Arch. Otolaryng. **15**:692 (May) 1932.

88. Ramadier, J.; Guillon, H., and Becker: Etude anatomique d'une voie d'abord des cellules de la pointe du rocher, Ann. d'anat. path. **9**:597, 1932.

is exposed. The carotid artery is exposed by removing bone between it and the tubal orifice. The exposure of the artery is extended toward its upward and horizontal portions. By means of temporary ligatures passed about it, the blood vessel is lifted out of its bed, and the petrous apex is entered through the inner wall of the bony carotid canal channel.

A critical analysis of the advantages and the disadvantages of these various procedures has recently been published (Kopetzky [89]).

There are numerous observations appearing in the current literature. Ramadier [90] reports 4 cases, in 2 of which the patients recovered. He details the cell structure of the pneumatized pyramid and divides the cells into two groups: (1) the intrapetrosal, including the infralabyrinthine (prefacial and subcochlear, precochlear or acute labyrinthine), and (2) the supralabyrinthine. A fistulous tract is apt to develop in tissue containing either of these types of cells. In these cases he did not use the technic already described.[88] In discussing the symptomatology, he gives a similar picture to that of Kopetzky and Almour. He ascribes the knowledge of the significance of the ocular pain to Ostmann (1897). Ramadier does not consider paralysis of the abducens nerve as part of the clinical picture of petrous suppuration. In the cases reported he used an operative technic closely resembling that of Eagleton.

Because the genesis of purulency of the pyramidal tract is still obscure, Brunner [91] reports in detail 2 cases with operative and pathologic findings. The cases came to autopsy because of a complication of lesions, including involvement of the tip. In one of the cases, among other findings there was a fistula into the carotid canal, and between the carotid canal and the bony part of the eustachian tube a thrombosis of the carotid plexus of veins had resulted. In the second case, autopsy revealed a break through the roof of the tympanotubal orifice. There was also a break into the gasserian fissure, with another into the carotid canal. Brunner discards the theory of paralysis of the abducens nerve as significant per se of disease of the pyramidal tip.

Lange [92] outlines cell tracts similar to those described by Ramadier, but under different names.

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89. Kopetzky, Samuel J.: Problems Concerned with Empyema of the Petrous Apex, Arch. Otolaryng. **18**:47 (July) 1933.

90. Ramadier, J.: L'ostéite profonde du rocher, Ann. d'oto-laryng., 1931, p. 1300.

91. Brunner, Hans: Ueber Eiterungen an der Felsenbeinspitze, Rassegna ital. di oto-rino-laring. **6**:221, 1932.

92. Lange, W.: Tiefgelegene Entzündungen im Schläfenzein, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:99, 1932.

Cases are reported, by Nager,[93] Eisinger,[94] Olsson[95] and Rosenblatt.[96] In most of these reports the clinical entity is not clear. There is confusion as to the so-called Gradenigo symptom, and in no case was a specialized surgical technic used. In Eisinger's case there were roentgen indications before operation. In Olsson's case an operation was followed by roentgen examination, which showed failure to reach the lesion. In a second operative attempt the lesion was reached, but the patient died. Postmortem examination showed a lesion corresponding to that seen in the x-ray films.

51 West Seventy-Third Street.

93. Nager: Zur operative Behandlung der Eiterungen an der Felsenbeinspitze (Gradenigoscher Symptomenkomplex), Versamml. d. Gesellsch. Schweiz. f. Hals- u. Ohren-Aerzte in Genf **6**:1, 1932.

94. Eisinger, K.: Gradenigoscher Symptomencomplex bei akuter Otitis: Mastoidectomy: Heilung, Osterreich. Otol. Gesellsch. Wien, 1932, p. 291.

95. Olsson, Y.: Un cas d'ostéite de la pyramide (du temporal) démontrée par la radiographie, Acta oto-laryng. **18**:163, 1932.

96. Rosenblatt, M.: Ueber einen Fall von Ophthalmoplegia contralateralis otogen Ursprungs, Zhur. Ushn., Nos. i Gorl. Bol. **9**:336, 1932.

Abstracts from Current Literature

Ear

TREATMENT OF CHRONIC SUPPURATIVE DISEASE OF THE MIDDLE EAR. J. D. McLAGGAN, Brit. M. J. **2**:94 (July 16) 1932.

Chronic otitis may follow a recurrent infection of the nasopharynx. It may be associated with a chronic infection of the mastoid antrum and air cells. In some cases the bulk of the disease lies chiefly in the region of the attic. In others the tympanum bears the brunt of the trouble. In still others there may be gross involvement of the entire cleft of the middle ear.

In the first group, in which otitis follows a recurrent infection of the nasopharynx, the usual history is one of recurrent discharge in childhood. This usually starts with a cold or sore throat. Pain in the ear quickly follows, which may be relieved during the first attack by rupture of the drum. In subsequent attacks the drum ruptures easily, and hence there is less pain. Treatment consists essentially in clearing up the infection in the nasopharynx, in addition to local treatment. The discharge is mucous or mucopurulent. In disease associated with the mastoid antrum, tenderness is elicited over the mastoid. The discharge is purulent. In addition to local care the important treatment is drainage of the antrum and of its air cells. Chronic otitis confined principally to the region of the attic is much rarer. It is more chronic. There is a history of aural discharge for years, without cessation and without much pain; the discharge is thin and watery. The attic must be laid open by an approach through the antrum. This operation is rather delicate. Hearing is preserved. In the fourth type, in which the disease is limited to the middle ear or to the tympanum, the otitis usually follows a recurrent infection of the eustachian tube, the source of which has been cleared up. Treatment by drops and by keeping the ear clean usually leads to a cure, but when this is not carried out ionization is successful. The middle ear and the external meatus are filled with a zinc sulphate solution after the ear has been thoroughly cleansed. Into this pool is inserted a metal electrode encased in vulcanite, which is attached to the positive pole. The negative pole is attached to a flat electrode placed on the patient's arm. A current of from 2 to 3 milliamperes is passed for from ten to fifteen minutes. This may require several repetitions. In massive infection of the middle ear there is usually a history of a profuse, purulent, frequently blood-stained and usually foul-smelling discharge. Acute attacks, with pain, headache and elevation of temperature, are common. There is tenderness over the mastoid. It is this type of infection which most frequently extends to the inner ear and the intracranial structures. Examination reveals gross destruction. A radical mastoid operation, with removal of the diseased structures in the middle ear, effects a cure, with loss of, or impaired, hearing in 70 per cent of the cases.

In all these conditions prophylaxis, general hygienic measures and care are important. FERGUSON, Niagara Falls, N. Y. [ARCH. NEUROL. & PSYCHIAT.]

OTITIS MEDIA IN SUCKLINGS. W. M. MOLLISON, Brit. M. J. **2**:581 (Sept. 24) 1932.

The author considers infants in the first year of life as sucklings and calls attention to the fact that the British literature on the subject of otitis media during the first year of life is rather scant. From the discussion, one would gain the impression that the examination of the ears with the otoscope as a routine measure is not common. Otitis media is classified as: (1) obvious otitis media,

with symptoms pointing to the ear; (2) latent otitis media, with no symptoms pointing to the ear, which includes (*a*) real otitis, discovered in infants apparently normal, and (*b*) pseudo-otitis, with general illness, crying, high temperature and refusal of food or with diarrhea and vomiting, or so-called parenteral disorders, and finally (3) tuberculous otitis media.

Symptoms and signs of obvious otitis media are outlined, and the treatment is described in more or less detail. Pneumococcic otitis media is emphasized, and its frequency is stressed. The author doubts that tuberculous otitis media occurs as often as has been indicated by some authors, but he states that when it does occur, it causes extensive destruction of the temporal bone with involvement of the glandular and facial nerve. When tuberculous otitis media is diagnosed, a thorough operation on the mastoid should be performed, and when this is done, there is a prospect of complete recovery.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

THE MENTAL EFFECT OF THE COMPLETE MASTOID OPERATION IN CHILDREN. MACLEOD YEARSLEY, Lancet **2**:613 (Sept. 17) 1932.

On the basis of the replies to a circular letter sent to directors of a number of schools for the deaf in England, Yearsley comes to the following conclusions concerning the mental status of children on whom complete mastoidectomy has been performed: There is good evidence that complete radical operation has a markedly deterrent effect on the education of the children. In the first place, there is loss of hearing, and in the second place, mental exhaustion and consequent inertia occur readily. The mental effect, apart from that produced by the toxemia of prolonged otorrhea, the author believes, is due probably to long continued pain together with fear of painful dressings. The condition is one of nervous exhaustion from prolonged suffering and mental strain, leading to irritability, emotional outbursts, lack of concentration, poor memory and want of balance in behavior. The effect is always more marked in children who have had to undergo more than one operation.

The accumulated data and conclusions are of interest, but it seems to the abstractor that insufficient account is taken of the defect in the eighth nerve as a psychopathologic stimulus. Furthermore, the possibility of distinct behavior defects due to fondling because of previous and prolonged serious illness is not considered.

BECK, Buffalo. [ARCH. NEUROL. & PSYCHIAT.]

NEW TRENDS IN THE THERAPY OF OTOSCLEROSIS. W. ROSENFELD, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:33 (Jan. 20) 1933.

In the opinion of Rosenfeld, otosclerosis is a localized form of osteomalacia. On the basis of this conception he combined the subcutaneous injection of small doses of epinephrine with the administration of calcium and cod liver oil in eighteen cases of otosclerosis. In all cases the tinnitus was greatly diminished or disappeared. In half of the advanced cases and all of the early cases the hearing capacity was considerably improved. The optimal dose of epinephrine varies with the individual patient, and a careful study of the reactions, comparison with other cases and much patience are sometimes required to determine it. The author found that the largest dose which was generally tolerated was 0.2 mg. He observed no injurious effects with this dose. He is convinced that the recognition of early cases of otosclerosis and treatment according to these principles will solve the therapeutic problems of otosclerosis.

EDITOR'S ABSTRACT.

SYMPATHETIC ANISOCORIA IN DISEASES OF THE MIDDLE EAR. RUTH WEYL, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:217 (March 21) 1933.

Weyl says that among thirty-five cases of destructive processes in the middle ear and radical operations on the middle ear there were nine cases of pronounced

anisocoria resulting from weakening of the sympathetic nerve on the side of the operation. In nine other cases there were constant differences in the pupils below the measurable size of 0.5 mm. Of these, only three cases exhibited signs of sympathetic paralysis; in six there was hyperexcitability. The clinical results indicate that radical operation on the middle ear in man, as in guinea-pigs, causes only partial destruction of the dilator trunk which passes the median tympanic wall, whereas in cats this nerve channel is entirely interrupted. The anisocoria is not the same in all cases because of individual anatomic variations and the concurrence of partial destruction and inflammatory irritation of scarred regions. In one third of the cases the pharmacologic reactions were those of a partial sympathetic paralysis; in the other cases the reactions could be attributed only to a combination of paralysis and stimulation. Paralysis of the sympathetic nerve is usually found in cases with a dry operative cavity, while stimulation of the sympathetic nerve was usually associated with a moist operative cavity or with an active disease process in the middle ear. In cases with concurrent paralysis and stimulation, the cocaine test clearly demonstrates the dominant component. The epinephrine test is less reliable. In the presence of differences in the facial nerve, such as follow the operation, individual components of Horner's syndrome, such as ptosis or the corneal reflex, cannot lose their diagnostic significance. Clinically the condition of the pupils after radical operation on the middle ear deserves attention because (1) otogenous anisocoria is more frequent than was previously assumed and (2) a change in the condition of the pupils indicates a change in the condition of the middle ear.

EDITOR'S ABSTRACT.

TOXICOSIS AND OTITIS MEDIA IN NURSLINGS. M. SALZBERGER and E. RABINOVICI, Monatschr. f. Ohrenh. **67**:188 (Feb.) 1933.

Salzberger and Rabinovici designate as toxicosis a nutritional disturbance occurring in infants in good health, usually in the first year of life. The condition is characterized by a sudden onset of vomiting and usually diarrhea, followed by apathy, loss of appetite and a rise in temperature. In severe cases the infants are stuporous or unconscious, the eyes are sunken, and the fontanels are depressed. The terminal stage is marked by tonic and clonic spasms and the appearance of coffee-ground vomitus. Toxicosis may have an alimentary, a parenteral or a mixed etiology. In Jerusalem it occurs mostly during periods of extreme heat and dryness. In eighty-two of the ninety-nine cases which form the basis of this report, otologic examination disclosed disease of the middle ear: in sixty-six cases suppurative otitis media, and in sixteen cases, catarrhal or simple otitis media. Paracentesis was performed in all cases with positive results. Recovery occurred in thirty-seven cases and death in thirty-nine; in six cases the result was unknown. In a series of cases in which nutritional therapy was without result, including several grave cases in which interruption of feeding and injections of dextrose had proved useless, paracentesis produced a sudden improvement in the general condition. In twenty-seven of the cases in which there was no improvement despite a flow of pus from the ear, mastoidectomy was performed. In all but two cases the mastoid process was pathologically changed, and in most cases it contained pus, but this was not under pressure. None of these patients had exhibited any clinical symptoms of mastoiditis. Nineteen of the twenty-three patients operated on died; twenty-three of the fifty-five patients not operated on died. The authors do not think that otitis media and possible suppuration of the mastoid process caused the toxicosis. They think that the unfavorable climatic conditions of heat and dryness favored the occurrence of a disease appearing under two chief manifestations: toxicosis and otitis media. Satisfactory drainage of the pus in the ear may often exert a favorable influence on the course of the disease, but otologic therapy must be accompanied by nutritional and general therapy. Mastoidectomy should not be performed as a routine measure, but only in cases of otitis media in which paracentesis did not effect an improvement.

EDITOR'S ABSTRACT.

CHANGES PRODUCED IN THE OSSEOUS LABYRINTH OF GUINEA-PIGS BY OVERDOSAGE OF VIOSTEROL. A. TOBECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:550 (March 20) 1933.

Tobeck reports that by overdosage of viosterol he was able to produce changes in the osseous labyrinth of guinea-pigs. These changes consisted, in the main, in the appearance of a marrow abundant in cells and containing giant cells, in an osseous decomposition through lacunar resorption and perforating canals, and in new osseous growth through the development of osseous trabeculae and margins. It may be assumed that the viosterol caused a disturbance of the calcium metabolism that resulted in the severe osseous changes. In view of the fact that the results of the latest investigations place otosclerosis among the large group of osteodystrophic bone diseases, the production of osseous changes in the otic capsule resembling otosclerosis by overdosage of viosterol is significant.

EDITOR'S ABSTRACT.

BONE CONDUCTION IN UNILATERAL DEAFNESS. H. WANGEMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:603 (March 20) 1933.

From a series of tests with the oto-audion on persons with unilateral deafness Wangemann concludes that stimulation of the labyrinth in bone conduction occurs via the osteotympanic as well as via the osseous route, at least for low tones. The relation of osteotympanic to osseous conduction shifts toward the osseous side with elevation in the pitch. In agreement with Weber's observations, a significant increase in bone conduction was noticed in plugging of the auditory passage. This increase from *C* 1 to *C* 3 is about tenfold. In cases of injury of the conduction apparatus of the ear, a decrease of bone conduction occurred when the ear was plugged. This shows that one must differentiate between a physiologic and a clinical Weber's test. The way the latter is commonly used it is only a modification of Schwabach's test. The results of this investigation indicate how the real Weber test could be made diagnostically valuable since it appears that in ears with impairment of sound conduction there is no improvement of bone conduction by plugging the ears, whereas this procedure results in a considerable improvement in ears with normal sound conduction. There should be a negative and a positive Weber test analogous to Rinne's test. A long series of experiments to determine whether the Weber and Rinne tests are always parallel would be necessary.

EDITOR'S ABSTRACT.

Pharynx

THE TONSIL PROBLEM. ARTHUR K. HOGE, West Virginia M. J. **29**:170 (April) 1933.

This article deals with the problem of tonsillectomy both in adults and in children. The results of tonsillectomy depend on the necessity for the operation and the careful selection of cases. Tonsils should not be removed during an acute infection or during an epidemic of acute disease of the respiratory tract, except possibly in an occasional emergency. Tonsillectomy should be performed only by those who are proficient in the examination of the throat, who are familiar with the anatomy of the area and who have had sufficient surgical training to enable them to meet any emergency that might arise. The method of operation is of little importance so long as removal is complete and there is a minimum of trauma. All points of bleeding must be ligated, and the patient must be kept under careful observation until recovery from the anesthetic is complete.

SHAWKEY, Charleston, W. Va. [AM. J. DIS. CHILD.]

INTRACUTANEOUS REACTIONS WITH TONSILLAR EXTRACTS. ERICH WIRTH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:4 (Jan. 20) 1933.

In the search for a method of determining whether a given general disease is related to the tonsils, Wirth experimented with intracutaneous injections of tonsillar extracts. He found that intracutaneous injections of tonsillar extracts produced early and late reactions of varying degrees. Some persons are hypersensitive to certain tonsillar extracts. No relations of diagnostic value could be found to exist between the intracutaneous reactions and the clinical pictures.

EDITOR'S ABSTRACT.

FOSSA SUPRATONSILLARIS. M. SASAKI, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:89 (Feb. 16) 1933.

Sasaki says that careful histologic investigation shows that the indentation of the tonsils usually called fossa supratonsillaris is always underneath the tonsillar capsule and has the same structure as the tonsillar crypts. As the term fossa supratonsillaris permits the belief that the fossa is superior to the capsule, the author suggests substituting the term fossa tonsillaris superior. The fact that the fossa is found in the tonsillar parenchyma has a clinical significance, and its structure must be especially considered in enucleation of the tonsil. The structure of this fossa also probably plays a large rôle in recurrent tonsillitis, peritonsillitis and its complications.

EDITOR'S ABSTRACT.

TONSILLITIS AND WEATHER. G. EIGLER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:119 (Feb. 16) 1933.

Eigler says that a study of the cases of tonsillitis at his clinic during one year showed that the number of cases increased during the months of seasonal change, during weather changes, during periods of constant high atmospheric pressure associated with a drop in temperature and during periods of low atmospheric pressure. Strong wind and storms also seemed to favor the development of the condition. About one half of the cases of tonsillitis appeared after operations on the upper respiratory tract, especially after septum resection. In fifty-seven cases in which submucous septum resection was performed, bacteriologic examinations showed a decided change in the flora of the pharynx and of the tonsils several days after the operation; the number of hemolytic streptococci in the epipharynx and the tonsils was greatly increased, and they were usually found abundantly in pure culture in the nose. Not all patients with hemolytic streptococci who were operated on had tonsillitis, but hemolytic streptococci were found in all patients in whom postoperative tonsillitis developed. In almost all of ten cases in which the opsonic index of the blood was determined, the patients showed an increased opsonic index for their own streptococci several days after the tonsillitis receded. These facts indicate that streptococci are the etiologic agents in postoperative tonsillitis. The epithelial cells of the tonsils and pharynx appear to have a phagocytic function; those of the nose do not. Examination of the pharyngeal mucosa before, during and after inflammation showed, besides the normal condition, pharyngitis granularis, diffuse cellular inflammation, edema and inflammatory fibrous hyperplastic conditions. The author concludes that the atmospheric conditions, the hemolytic streptococci and the existing condition of the pharyngeal mucosa before intervention are the determining factors in postoperative tonsillitis.

EDITOR'S ABSTRACT.

CHRONIC TONSILLITIS AS THE ETIOLOGIC FACTOR IN POSTOPERATIVE COMPLICATION IN GYNECOLOGY. M. E. HINDES and W. A. POWSCHITKOW, Monatschr. f. Ohrenh. **66**:1427 (Dec.) 1932.

Hindes and Powschitkow call attention to chronic tonsillitis as a source of postoperative complications. They conducted necropsies on seven of fourteen

unexplained postoperative deaths constituting part of a total of eighty-five deaths which occurred among 10,256 gynecological operations. In four of these seven cases, suppurating foci were found in the tonsils which must be regarded as the source of the septic peritonitis and septicemia which caused death. In the other three cases, the origin of the septic peritonitis could not be ascertained. The authors think that all patients should be examined prior to operation, in cooperation with the laryngologist and the internist. When chronic tonsillitis is present in a patient requiring surgical intervention, healing of the diseased tonsils should be effected first. In all cases of postoperative death of unexplained origin, postmortem examination of the tonsils is recommended.

EDITOR'S ABSTRACT.

INFLAMMATORY COMPLICATIONS OF THE WOUND FOLLOWING TONSILLECTOMY. N. SIEGMANN, Monatschr. f. Ohrenh. **66**:1458 (Dec.) 1932.

Siegmann discusses the literature and reports twelve cases that occurred in Hajek's Clinic between 1926 and 1931 and a few others. The series comprises: five cases of inflammatory infiltration of the wound; one case of pneumonia with inflammation of the wound, followed by otitis; four cases of peritonsillar abscess; four cases of parapharyngeal or retropharyngeal abscess, and one case of infiltration toward the mediastinum. The author thinks that these postoperative inflammatory complications can probably be attributed to faulty anesthetizing technic, consisting in an injection into diseased adenoid tissue or the carrying of bacteria from the surface of the mucosa into the submucous tissue with the hypodermic needle. By injecting too deeply one may cause local infection either of the pharyngeal musculature or of the parapharyngeal space (by piercing the pharyngeal wall), with danger of spread in several directions, particularly toward the mediastinum. The author emphasizes the importance of waiting four weeks after the last attack of tonsillitis.

EDITOR'S ABSTRACT.

A CLINICAL AND PATHOLOGICO-ANATOMIC CONTRIBUTION TO THE CLONIC, RHYTHMIC CONTRACTIONS OF THE PHARYNGEAL MUSCULATURE. W. SCHMITT, Nervenarzt **5**:456 (Sept. 15) 1932.

After considering the clinical features of clonic, rhythmic contractions of the pharynx, Schmitt enumerates the lesions found at autopsy in previously reported cases. He is particularly impressed with the finding, in some cases of unilateral spasm, of a lesion in or lateral to the dentate nucleus on the same side, with an associated atrophy of the corresponding brachium conjunctivum and the opposite red nucleus and inferior olive. In the case here reported, the findings were suggestive of a left cerebellar lesion when the patient was first seen. On reexamination three months later, very rapid rhythmic movements were seen in the left soft palate and left faucial pillars, as well as in the musculature of the left side of the pharynx. Nothing was found on exploration of the posterior cranial fossa two months after this, but at the time of a second surgical attack nearly a year after the patient was first seen, a large mass of neoplastic tissue was removed from the left cerebellar hemisphere. The patient died, and at autopsy no changes were found either in the right cerebellar hemisphere or in the basal nuclei, pons or medulla oblongata.

DANIELS, Denver. [ARCH. NEUROL. & PSYCHIAT.]

PRIMARY TUBERCULOSIS OF THE TONSILS. H. HAMPERL and K. WALLIS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:480 (Feb. 12) 1933.

In connection with a case of primary tuberculosis of the tonsils, confirmed by necropsy, in a nursling aged 3 months, Hamperl and Wallis point out the difficulty of the diagnosis of this condition from the anatomicopathologic and especially the clinical standpoint. Cases in which an unquestionable diagnosis of primary tuber-

culosis of the tonsils can be made are extremely rare. In the case described, the mother of the child had active pulmonary tuberculosis, with many acid-fast bacilli in the sputum. It is probable that the inoculation of the tubercle bacilli occurred through the mother's practice of moistening the nipple of the nursing bottle with her own saliva.

EDITOR'S ABSTRACT.

Larynx

NONDIPHTHERITIC LARYNGOTRACHEOBRONCHITIS. S. KIRKPATRICK and S. M. KIRKPATRICK, South. M. J. **26**:287 (March) 1933.

The predominating organisms found in laryngotracheobronchitis are staphylococci and streptococci. Low tracheotomy with mechanical removal of mucus is essential in severe cases. Fluids should be forced. Antispasmodics are also useful. Laryngotracheobronchitis is a serious disease, with a mortality around 50 per cent, and it is often diagnosed as diphtheria. The condition is much more prevalent than was formerly thought. An early diagnosis should be made and energetic treatment instituted.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

FUNCTIONAL APHONIA: SOME OBSERVATIONS ON ITS CLINICAL ASPECTS. F. DOUGLAS MARSH, Lancet **2**:289 (Aug. 6) 1932.

Functional aphonia is an inability to produce the voice owing to failure of the neuromuscular apparatus in the absence of organic disease. A summary of sixteen cases shows that functional aphonia may be associated with sepsis in the upper air passages. Functional aphonia has been synonymous with hysterical aphonia. The author suggests that it is advisable to classify functional aphonia into two classes, primary and secondary. The primary class is exemplified by the purely hysterical forms, and the secondary class by the forms that start with laryngitis secondary to a septic focus in the upper air passages. The series of cases reported shows clearly that a determining cause is often present. When found, it should be treated and should be regarded as no less important than the restoration of the voice. There is seldom any difficulty in obtaining complete restoration of the voice by electrical stimulation, by suggestion or by instruction in proper voice production. This, with proper handling of the septic focus, will do much to prevent relapse, which otherwise is apt to be frequent.

BECK, Buffalo. [ARCH. NEUROL. & PSYCHIAT.]

THE NEURAL APPARATUS OF THE POSTERIOR CRICO-ARYTENOID MUSCLE IN MAN. P. SUNDER-PLASSMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:586 (March 20) 1933.

In studying the neural apparatus of the posterior crico-arytenoid muscle, Sunder-Plassmann was struck first of all by the coarse structure of the terminations of the motor nerves and by the abundance of nerve tissue, which absolutely, as well as relatively, exceeds that of the other laryngeal muscles. The ramifications of the recurrent nerve spread through the entire muscle and can be traced under rather low magnification to their terminations. He thinks that the unusual size and abundance of the motor nerve terminations (sometimes several were seen on one muscle fiber) serve to carry every nerve impulse rapidly to the abductor of the glottis. The bifurcate and multiple terminations represent the real ends of the motor recurrent fiber, around which the nuclei of the motor end-plate are grouped. The nerve endings are connected with these nuclei by neural protoplasmic connections like a periterminal network.

The posterior crico-arytenoid muscle contains many sympathetic ganglions and many of these are along the main trunk of the recurrent nerve. Also single ganglion cells appear frequently along the branches of the recurrent nerve. The fine sympathetic nerve fibers which issue from the ganglion cells do not have definite termina-

tions but form a large, loose-meshed and unusually delicate terminal reticulum which spreads over the entire muscle and appears to pass into the sarcoplasm of the striated muscle fibers. It can be seen only under high magnification. It may correspond to that described by Stöhr in the gastro-intestinal tract and by Reiser in the appendix. This terminal reticulum spreads over or through the capillaries, the fibers of the musculus vocalis and the mucous glands of the vocal cords. The author regards it as the neural basis for tonic-trophic influences on the musculature. Another type of nerve termination, seen in slender nerve fibers with finely granulated protoplasm, consists in a fine protoplasmic network passing into the sarcoplasm of striated muscle and surrounding finely granulated large nuclei; these the author assumes to be accessory sympathetic nerve endings, possibly the basis for the sympathetic muscle tone.

EDITOR'S ABSTRACT.

Nose

SINUS DISEASE IN CHILDREN. V. W. FISCHBACH, West Virginia M. J. **29**:145 (April) 1933.

The prime object of this article is to emphasize the importance of careful and thorough study of the nasal sinuses of children with symptoms pointing to involvement of the head. Sixty-five per cent of the patients with chronic sinus infection give histories of repeated acute infections of the head in infancy. The remaining 35 per cent present histories showing contributory causes. Many patients with otitis media and with frequent colds are not relieved by removal of the tonsils and adenoids. Many cases of more general systemic infections are due to focal infections in the nasal sinuses. The primary cause of sinusitis is infection, but allergy, dietary factors and contact with chronic cases may be contributory causes. Examination, symptoms, diagnosis and treatment are discussed in detail.

SHAWKEY, Charleston, W. Va. [AM. J. DIS. CHILD.]

ANATOMICOCLINICAL STUDY OF POSTTRAUMATIC NASAL CAUSALGIA WITH PUPILLARY DISTURBANCES. J. CREYSSEL and P. MOUNIER-KUHN, Rev. d'oto-neuro-opht. **10**:663 (Nov.) 1932.

A man, aged 33, suffered a severe wound of the left side of the nose and face. Following the accident there developed a painful vasomotor and secretory syndrome, with nasal pain, tumefaction and congestion, obstruction and rhinorrhea, appearing in crises from various causes, principally exposure to cold; a syndrome of alternating vasodilatation in the turbinates of each side of the nose, with concomitant pupillary modifications—mydriasis on the side of the vasoconstriction and myosis on the vasodilated side, and, later, a typical causalgic syndrome, with violent superficial pain, occurring by crises after the least touch on the nose and relieved by pressure. The scar, at first normal, became edematous with a white spot in the middle, which was hypersensitive and seemed to be the center for neuralgic radiations. After the cicatrix and the middle turbinate body had been removed and the nose had been treated, the symptoms disappeared. Histologic examination of the excised scar did not reveal manifest nerve lesions. The explanation of the symptoms therefore rests on vasomotor disturbances that brought about in the fibrous tissue intermittent compression of the nerve filaments in a richly innervated area. Therapy consists in removing the irritating focus and, if necessary, the superior cervical sympathetic ganglion.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

RETROBULBAR NEURITIS OF NASAL ORIGIN. A. FEIGENBAUM and M. SALZBERGER, Folia oto-laryng. orient. **1**:50 (Oct.) 1932.

The authors report twenty-four cases of retrobulbar neuritis. There were nineteen males and five females, and the disease occurred between 13 and 67 years of age, the average age being 33½. Seventeen cases were bilateral. Many of

these cases occurred about four weeks after an epidemic of influenza. The fields usually showed central or paracentral scotomas. The blind spot was enlarged in two instances. Examination of the fundus was negative in fifteen; there was pallor in seven, papillitis in one and a small hemorrhage, together with narrowed arteries, in the remaining case.

Nine cases showed definite sinus disease, while the roentgenograms were positive for sinus infection in twelve cases. Multiple sclerosis could be ruled out in all cases.

Since conservative treatment seemed ineffective, the sphenoid and posterior ethmoid cells were opened in sixteen cases. Microscopic or macroscopic pathologic changes were found in all these cases. Eight of the patients who were operated on and three on whom operation was not performed were cured.

The authors feel that an inflammatory edema plus a direct toxic effect (on the papillomacular fibers) are the causal factors. The fact that most of these cases followed influenza is offered as a reason for considering that the etiology was nasal in this particular group of cases.

DUGGAN, New York. [ARCH. OPHTH.]

BACTERIOLOGY AND HISTOLOGY OF THE ACCESSORY NASAL SINUSES. W. JENSSEN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:439 (Feb. 12) 1933.

Jenssen reports on a bacteriologic and histologic study of the nasal sinuses of forty skulls. A little more than half of the sinuses which were histologically normal were sterile. The frontal sinus was most frequently sterile. The flora is relatively uniform in comparison with the typical nasal flora. The conditions of examination could be considered as physiologic, since postmortem invasion of the nasal sinuses does not start until relatively late. A specific flora of the sinuses comparable to the specific flora of the distal part of the appendix and the specific flora of the tonsillar crypts does not exist. The author describes intravital and especially postmortem changes and shows how they may be differentiated from other processes. The phenomenon of elective phagocytosis usually permits identification of the etiologic agent among the organisms found in each case. The chronic serous inflammation of the nasal sinus develops on the basis of a chronic edematous condition of the mucosa characterized by functional deficiency. The recurrences are produced by an increase in the virulence of the saphrophytic inhabitants of the sinus and a decrease in the resistance of the mucosa occurring in so-called "colds" or general infection, not through new infection with pathogenic organisms from the nose. In cases of extensive polyposis the inflammation can be caused by accumulation of secretions. A certain uniformity in the bacteriology of acute infection of the nasal sinuses and the tonsils appears indicated by the predominance of hemolytic streptococci.

EDITOR'S ABSTRACT.

ACCIDENTS IN LOCAL ANESTHESIA OF THE SEPTUM. E. ZIEGLER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:476 (Feb. 12) 1933.

Ziegler reports that during the injection of a 2 per cent solution of procaine hydrochloride with epinephrine into the left side of the septum, the patient suddenly complained of scintillation and inability to move the left eye. Simultaneously complete anemia appeared in the skin on the left half of the nose and in a strip across the forehead from the median line laterally to the middle of the eyebrow and upward to the hair. No difference between the fundi oculi of the left and right eyes could be seen. The anemia disappeared toward the end of the operation, which was performed without further mishap, and the subjective ocular disturbances also disappeared. As the amount of procaine hydrochloride injected was far below the toxic dose, the author thinks the epinephrine must be regarded as the cause of the accident. Injections of epinephrine may cause neural, respiratory or circulatory disturbances in persons with hyperirritability of the sympathetic system. The patient belonged to this group. The proximity of the manifestations to the site of injection should not be taken as a sign that the epinephrine accidentally penetrated the blood stream and was conveyed directly into the supra-orbital and

angular arteries and the dorsal artery of the nose; rather, the irritation produced by the epinephrine at the site of injection was conveyed by a nervous impulse to the regions in which the vascular contraction occurred. The mechanism is unknown, but the sympathetic plays a major rôle.

EDITOR'S ABSTRACT.

OPERATION ON THE ACCESSORY NASAL SINUSES, ESPECIALLY IN PANSINUSITIS. E. MATIS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:527 (March 20) 1933.

Matis describes a new method for surgical therapy of pansinusitis from the vestibulum nasi which is accomplished without leaving facial scars. The method is indicated in cases in which one wishes to proceed more conservatively than in external, but more radically than in intranasal, operation. After making an incision of the apertura piriformis, which should be as long as possible, the soft parts of the region of the facial wall of the maxillary sinus and the orbital wall of the frontal sinus are extensively detached. For operation on the maxillary sinus, Matis uses Sturman's method; for the other sinuses he uses his own technic. The frontal sinus is opened at Kuhnt's point after pushing aside the soft parts over the anterior lacrimal crest. After removal of the floor of the cavity the sinus may be cleaned out as much as necessary. The resection of the frontal process of the superior maxilla — the technic is thoroughly discussed — creates not only a wide passage through the frontal sinus but a convenient access to the ethmoid cells. The size of the resection must be varied according to the anatomic conditions. Generally the upper part of the frontal process must be resected extensively and the lower part sparingly. In difficult cases and in those with orbital complications the frontal process and the lacrimal bone are radically extirpated. In connection with the cleansing of the ethmoid cells, the operation on the sphenoid sinus is performed. The author points out the possibility of exposing the other sphenoid sinus.

EDITOR'S ABSTRACT.

Miscellaneous

ANLAGE AND PNEUMATIZATION. W. ALBRECHT and M. SCHWARZ, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:50 (Jan. 20) 1933.

Albrecht and Schwarz reply to Wittmaack's article on the constitution of the mucosa and pneumatization (*Arch. f. Ohren-, Nasen- u. Kehlkopfh.* **132**:261 [Aug. 23] 1932; abstr., ARCH. OTOLARYNG. **17**:585 [April] 1933) in which he said that genotypic anlage cannot be recognized in the morphologic picture but only in the biologic reaction of the various tissues and contradicted the conclusions reached by the authors in a comparative study of the mucosae of 145 fetuses and newly born infants. They found that idiotypic variations in the structure of the connective tissue matrix of the mucosa can be demonstrated not only in the middle ear but also in the accessory nasal cavities. These individual variations are due to a variable tendency of development of the fibroblasts and an associated tendency toward fibril formation. Wittmaack regards these variations as postmortem changes, and stated that he was able to produce variations in the morphology of the mucosa identical with those described by the authors, by varying the manner and duration of preserving the specimen before fixation. The authors are convinced that postmortem changes played no rôle in the variations seen by them, as they exercised the utmost caution in that respect. They think that the morphologic variations seen by Wittmaack were due to the acceleration of postmortem changes produced by placing the specimens in bouillon and in the incubator. This results in a swelling of the tissue, especially the collagenic fibers. Under oil immersion these conditions of swelling can be clearly distinguished from the stages of development described by the authors. Postmortem changes can be recognized, and they cannot result in a change in the nature of the mucosa. The authors consider as highly improbable Wittmaack's theory that a single inflammation during the nursling age could change the entire character of a healthy mucosa and determine the type of bone formation in the mastoid process. They do not consider the

compact mastoid process as a disturbance in development but as an anomaly, determined by the genotypic anlage as are the other grades and forms of pneumatization. They admit, however, that inflammation may disturb the pneumatization in case of a constitutional inferiority of the tissue. EDITOR'S ABSTRACT.

TREATMENT OF ACTINOMYCOSIS. A. BREMSER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:94 (Feb. 16) 1933.

Reviewing the literature which has appeared in the last decade on the therapy of actinomycosis, Bremser found that roentgen rays, potassium iodide or a combination of roentgen rays and potassium iodide gave the best results. Potassium iodide alone was employed the least and gave the smallest percentage of cures; the combination of roentgen rays and potassium iodide was employed most frequently and gave the largest percentage of cures. The author reports eleven cases of actinomycosis from his clinic. Of ten patients with cervicofacial localization, six were treated with high voltage roentgen irradiation and four with a combination of roentgen irradiation and potassium iodide. One patient with actinomycosis of the tongue received only potassium iodide and arsenic. All but two patients still under treatment were cured, and examinations made from one and one-half to twelve years after treatment showed no recurrences. Although the results obtained with high voltage roentgen irradiation were just as good and as lasting as those obtained with the combination of roentgen rays and potassium iodide, the author thinks that potassium iodide should not be discarded entirely as a medicine for actinomycosis as it seems to exert a favorable influence on the healing process of this disease as shown in the case of lingual actinomycosis. The best treatment is high voltage roentgen irradiation, and the best results are obtained by giving large doses from the outset. In the author's opinion, the maximum dose is the optimal dose. EDITOR'S ABSTRACT.

SPECIFIC DESENSITIZATION THERAPY OF HAY FEVER AND NONSPECIFIC DESENSITIZATION THERAPY OF RHINITIS VASOMOTORIA. BELA SZENDE, Monatschr. f. Ohrenh. **67**:39 (Jan.) 1933.

Szende reports favorable results with desensitization by means of injections of a polyvalent pollen allergen in fourteen patients with hay fever who gave positive cutaneous reactions to this allergen. After a graduated series of thirty injections at two day intervals has been given, he continues to inject one ampule a month for a year. If a reaction occurs before the series has been completed, the injections are discontinued for a few days and later injections of the maximum tolerated dose are resumed at weekly, and then at monthly, intervals. While it is best to give the injections before the hay fever season, one patient with active hay fever was relieved from symptoms after four injections. In two cases of hay fever combined with asthma, desensitization was accomplished in one case by a mixed mold-fungi allergen and in the other case by a house dust allergen; both patients had given positive reactions with the allergen but negative reactions with pollen extract. In the treatment of vasomotor rhinitis with a nonspecific protein antigen, the author used a vaccine of tuberculin, staphylococci, streptococci, pneumococci and influenza bacilli which was rubbed into the skin after scarification; six applications were made. In all but five of twenty-six cases, the frequency and intensity of the attacks were greatly decreased (in one case the interval between attacks was as long as six months), but no permanent cures have been obtained. This treatment is contraindicated in active tuberculosis. EDITOR'S ABSTRACT.

SCLEROMA OF UPPER RESPIRATORY PASSAGES. A. PUTSCHKOWSKI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:265 (Dec. 22) 1932.

Putschkowski reports some observations made on 700 clinical cases of scleroma. His own experience and that of other Russian clinicians contradicts the opinion that scleroma and tuberculosis rarely occur simultaneously. Quite a few of his

patients with scleroma had tuberculosis of some other organ, in most cases tuberculous cervical lymphadenitis. In adult patients with scleroma and pulmonary tuberculosis, the tuberculosis almost always assumed a fibrous form and its evolution was favorable. This observation was also made by Sendulski. When tuberculosis and scleroma (particularly of the larynx) occur simultaneously in young persons, the prognosis is less favorable and the tuberculosis may terminate fatally. Scleroma of the trachea without involvement of the larynx, rarely reported in the literature, was observed by the author in five cases, and scleroma of the bronchi with scleroma of the larynx but an apparently normal trachea was observed in nineteen cases. In these cases the scleromatous process consisted in hard infiltrations, rather than in nodose proliferations. In two of the nineteen cases there was also pulmonary inflammation; both ended fatally. Necropsy disclosed the pneumonia as an unusual type of catarrhal desquamative process with lymphocytic reaction. While diseases of the eustachian tube and of the middle ear frequently result from partial or total occlusion of the tube by scleromatous nodules or infiltrations, the spread of the scleromatous process from the nasopharynx to the eustachian tube and the middle ear has been reported only twice. The author saw two cases in which the scleromatous proliferation filled the eustachian tube: in one it involved also the middle ear, the tympanic membrane and the external auditory meatus.

EDITOR'S ABSTRACT.

MUCK'S EPINEPHRINE PROBE TEST. E. WIRTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:381 (Dec. 22) 1932.

Wirth says that the results obtained with Muck's epinephrine probe test in 166 patients with otosclerosis, diabetes, migraine, epilepsy, Ménière's disease, cerebral syphilis, tumor of the brain, fracture of the skull and apoplexy did not differ significantly from the results obtained in 161 normal persons. Negative reactions were found in 68 per cent, questionable reactions in 25 per cent and positive reactions (white streak sign) in 7 per cent of those examined. The white streak sign is observed chiefly in persons in whom the mucosa of the inferior concha is characterized by a cushion-like swelling. The nature of the mucosa seems to be important in the production of a positive reaction. The author's experiences do not indicate a relationship between the white streak sign and injury of the meninges.

EDITOR'S ABSTRACT.

TERTIARY SYPHILITIC CHANGES OF SKULL. C. VON EICKEN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:395 (Dec.) 1932.

Von Eicken reports a case of tertiary syphilitic changes of the skull in a man aged 35. The clinical symptoms started with rhinitis and formation of crusts diagnosed as syphilitic ozena with extensive perforation of the septum and partial destruction of the medial wall of the maxillary sinus. The larynx exhibited swelling, scars and masses of granulation tissue. A swelling which started between the eyebrows gradually extended to the parietal bone; on pressure, pulsation could be felt, and it appeared that a piece of the frontal bone the size of a silver dollar was lacking. A similar swelling appeared on the left tuber frontale. The patient showed great weakness and marked loss of weight. The roentgenogram showed zones of greatly reduced density in the frontal and parietal bones; the frontal sinus itself was shadowed; the nasal cavity was unusually wide, and the conchae were largely destroyed. Necropsy disclosed severe tertiary syphilis with extensive destruction of the front half of the cranial vault extending to the bones of the face and also syphilitic scar formation in the larynx and generalized amyloidosis.

EDITOR'S ABSTRACT.

Society Transactions

SECOND INTERNATIONAL CONGRESS OF OTORHINOLARYNGOLOGY

Madrid, Spain, Sept. 27-30, 1932

M. G. Tapia, M.D., *President, in the Chair*

Pathologic Anatomy and Pathogenesis of Otosclerosis. Dr. Otto Mayer, Vienna, Austria.

The osseous neoplasm in otosclerosis is resorptive, consists primarily of endochondral bone and develops in the cavities and arches of the otic capsule in the form of foci or cords which surround this area in ringlike formation. It is classified under tumorous hyperplasia because of the construction and inclination to hyperplasia. A small accumulation of endochondral tissue at the point of origin of the neoplasm presents as a type of tissue malformation, from which the neoplasm develops, the hamartoma of E. Albrecht and the hamartoplasia of M. Weber. The neoplasm originates in the margins of the fenestra and the arches of the osseous capsule of the labyrinth. From the standpoint of structure this development is strongly mechanical. Fissures are found in the same places as a result of mechanical taxation of the bone. Thus the osseous neoplasm in otosclerosis must result from the mechanical taxation. This is all the more probable since similar pathologic processes in the bones are well known. Accordingly the osseous neoplasm in otosclerosis should have a mechanical-functional history. It serves to build up a support in an area of especially great tractional force. The hyperplasia and the ankylosis of the stapes, according to this interpretation, appear as the result of a tendency of nature to excessive regeneration. The neoplasm may then be interpreted as a callus-like formation and the entire process as a constant callus formation similar to that thoroughly described by Looser in the so-called zones of the transformation (*Deutsche Ztschr. f. Chir.* **189**:113, 152 and 189, 1924). Besides the mechanical-functional taxation, the constitutional factors in the development cause a local or general inferiority or oversensitivity of the bone to irritation of any sort. Possibly other general factors enter into this as the cause of a general inferiority of the bone, of which nothing definite is known.

It may be concluded that the osseous new growth in otosclerosis results from a mechanical-functional taxation, due on the one hand to disproportion between the mechanical taxation and the capacity of resistance and on the other hand to an unusual congenital capability of reaction to mechanical irritations. The purpose of the osseous neoplasm is the mechanical protection of the fenestra, the inner auditory meatus and the semicircular canals of the otic capsule, which are especially imperiled by the tractional tension. The protection takes place through the infiltration of an unusually resistant bone tissue instead of the mechanically inferior bone.

DISCUSSION

Dr. Neumann: The histologic picture of otosclerosis remains vague. Two views are held regarding the manner in which the disease begins: (1) that a resorption of the osseous labyrinth precedes the development of otosclerotic foci and that the areas of resorption are followed by a new deposition of osseous deposits and (2) that degenerative alterations of the osseous labyrinth take place accompanied by secondary processes of transformation (Wittmaack, Eckert-Moebius, Döderlein, Lange). Politzer was one of the first to consider otosclerosis a primary disease of the labyrinthine capsule. He differentiated between the malignant and

the benign form; in the former there are scarcely any osteoclasts, and in the latter there is active resorption. Otosclerosis is more frequently diagnosed than it exists; were that not the case, sections showing histologic evidence of otosclerosis would not be so rare as they are.

Dr. Quix: Clinical reports on otosclerosis show that only the auditory organ becomes infected; the vestibule remains intact, while alterations, mainly of the fenestra, are found in the capsule of the canals and vestibule. It is not generally understood that if the osseous alterations were the only cause of degeneration of the auditory organ the function of the vestibule would remain intact. The osseous alterations are secondary. The symptomatology of otosclerosis consists in a defect of sound transmission through the ossicles in a disorder of the internal ear or in both.

Dr. Portmann: According to Dr. Mayer, the secondary fissures in the osseous labyrinth are filled with callus. In cases of fracture or of fissures secondary to traumatism, may the callus stopping these fissures be the basis of otosclerosis? In a case of fissure or traumatic fracture of the petrous portion of the temporal bone, followed after several months or years by deafness of otosclerotic type, can a cause and effect relationship be assumed?

Dr. de Parrel: The following deductions were made from observations of deaf children. Pure otosclerosis, with intact tympanums and eustachian tubes, prolonged osseous conduction, a negative Rinne test, elevation of the inferior limit, paracusis of Willis and ringing, is not the most frequently observed type. There seem to be many more mixed and secondary cases. False otosclerosis includes such processes as congenital syphilitic or familial labyrinthosis and otoneurosis without the characteristic lesions of the otic capsule. Heredity enters into one half of the cases of pure or mixed otosclerosis. All the patients observed presented (1) symptoms of intestinal stasis and hepatocolic autointoxication and (2) dysfunction of the venous circulation with edema, varicose veins and an inclination to rest, and there was (3) endocrine dysfunction in many young patients. Opotherapy and intestinal disintoxication proved valuable. For the treatment of humming in the ears, diathermy was administered, together with sedatives acting on the nervous system. Lip reading has been of great service to otosclerotic patients.

Dr. Maurice Sourdille: On the basis of 150 operations on 67 ears, of which 62 presented a clinical syndrome of otosclerosis with osseous ankylosis of the stapes, the following observations were made:

1. A permanent fistula on the external semicircular canal or its ampulla slackens or even stops the progress of deafness on the operative side. Inversely, if the labyrinthine fistula is closed by an osseous callus, the progress of the deafness is restored.

2. Only the permanence of the labyrinthine fistula is to be taken into account in these modifications of the evolution of deafness.

3. The labyrinthine fistula causes not only an improvement in hearing on the operative side but a temporary improvement on the other side.

4. On opening an otosclerotic labyrinth in which an ankylosis of the stapes has been controlled operatively, one finds that very good hearing—from 8 to 10 meters for a low voice—is compatible with important osseous lesions.

Dr. Kelemen: Personal observations revealed that 14 series of horizontal sections from fractures of the base of the skull with a fatal outcome and involvement of the petrosa were without fissures—a sign that serious traumas do not necessarily cause fissures.

Dr. Bilancioni: Fetal pathology has great importance in otologic pathology. There are congenital, familial and hereditary forms, ostensibly due to osseous and articular forms which concern the osseous architecture of the ear, and which are probably due to alterations of the thymus, the parathyroids and the sexual glands.

Dr. Otto Mayer: Drs. Portmann and Kelemen brought out an important point. That traumatic fractures of the base of the skull do not always lead to

otosclerosis is not to be marveled at simply because the constitutionally specified abnormal reaction of the osseous tissue must lead to mechanical irritation. That this abnormal reaction can be brought on by a disturbance in metabolism was established by Weber, who showed that in animals with a diet deficient in calcium a circumscribed fibrous osteitis and a brown tumor can appear on the maxilla. The general disturbance in metabolism predisposes to the appearance of circumscribed osteitis fibrosa in a mechanically irritated spot like the maxilla. One might produce otosclerosis by creating a disturbance in metabolism and then mechanically and traumatically irritating the capsule of the labyrinth. Thus otosclerosis may appear after fracture of the base of the skull. Dr. Bilancioni's reference to the possibility of endocrine disturbance cannot be disproved. The most important lines of study lie in general osseous pathology. Ernst says that every formation depends on function, and that the function of the bone tissue is mechanical. The process of otosclerosis is not unusual; it not only occurs in the capsule of the labyrinth but is analogous to other processes of the bone system. If research is continued along these lines, the clarification of the problem of otosclerosis will be only a matter of time and zeal.

THE CLINICAL ASPECT OF OTOSCLEROSIS. DR. J. S. FRASER, Edinburgh, Scotland.

One can distinguish five types of otosclerotic patients: 1. Those with normal drum heads and eustachian tubes but suffering from progressive deafness with or without tinnitus and paracusis. On functional examination they give the well known Bezold triad in tuning fork tests. 2. Those with similar symptoms but with slight opacity of the drum heads. 3. Those with the upper tone limit depressed and the lower tone limit raised, a negative Rinne test and some loss of bone conduction (advanced cases with mixed middle and internal ear deafness). 4. Those with secondary sclerosis, suffering from progressive deafness with or without tinnitus or paracusis; (*a*) those whose drum heads show the result of inflammation of the middle ear; (*b*) those with suppuration of the middle ear (microscopic examination in a number of such cases has demonstrated spongification of the labyrinthine capsule in addition to the suppuration of the middle ear; (*c*) those with obstruction of the eustachian tube and changes in the drum head indicative of catarrh of the middle ear. 5. Those under 35 who have normal drum heads and eustachian tubes but suffer from progressive deafness with or without tinnitus or paracusis and have a family history of deafness (atypical otosclerosis). The British Otological Committee reported that 44.4 per cent of patients with otosclerosis have a family history of hereditary deafness. Otosclerosis occurs more frequently in females. Exposure to severe cold, anemia, rheumatic and focal infections, endocrine disorders, fragility of the bones, blue sclera and arteriosclerosis were cited as conditions associated with the disease. British otologists found that a majority of patients suffering from otosclerosis complained of tinnitus in such forms as the sound of steam from a kettle, the sound of a sea shell, whistling, the ringing of bells, the sound of wind in the trees and humming. The tinnitus was most often localized in the ear and next often in the head. Only a little over 50 per cent of the patients had paracusis. In most cases the eustachian tube was patent. According to this committee, the degree of deafness in otosclerosis corresponds to the raising of the lower tone limit: for example, in cases of slight deafness only the lowest forks failed to be heard by air conduction, while in very marked cases the first fork heard might be *C* 256 or *C* 512. Ninety-two per cent of British physicians had never found a case of otosclerosis resulting in complete deafness. Most of the time iodides, strychnine and preparations of iron, arsenic, phosphorus and calcium were the remedies used by otologists; sodium salicylate, cod liver oil and thiosinamine were seldom used. Strychnine proved to be most beneficial. Bromides and phenobarbital were favored in the treatment of tinnitus. Endocrine preparations, vaccine therapy, ultraviolet radiation, otomassage and electrical treatment

gave good results in a few cases. Operations on the nose met with little success. Ear trumpets, the long hearing tube and celluloid auricles were found of service in producing artificial hearing. Considerable reliance was placed on lip reading.

SOME OF THE WORK IN PROGRESS IN AMERICA ON OTOSCLEROSIS: EMBRYOLOGIC, METABOLIC AND GENETIC. Dr. J. GORDON WILSON, Chicago.

The research of a committee of the American Otological Society reveals that no marked improvement in hearing can be expected in any case in which the disease process has existed for much more than a year. On the other hand, when otosclerosis has not existed for more than a year and when the amount of bone change, as judged by the hearing test, is not great, an improvement may be effected. In three cases, none of more than twelve months' duration, the improvement was marked; the hearing became normal (audiometer reading) though the tinnitus did not quite disappear. This improvement has been maintained for two and a half years. In older patients in whom the process is of longer duration, a slowing of the progress (which possibly represents a cessation of the disease) may be expected to result from a diet featuring a diminished calcium intake. Such a diet is easy for any patient to carry out. While the use of parathormone to remove calcium from the body greatly assists in increasing the calcium output, its use has been found unnecessary. The extract is expensive, and the patient must be hospitalized. The same result can be obtained in a simpler, slower way by the oral administration of from 50 to 60 Gm. of ammonium chloride, the amount being limited to the patient's tolerance. A delayed advance may be obtained but no resolution. These findings may be regarded as tentative. In the genetic study of otosclerosis, the hypothesis that otosclerosis depends on a single dominant factor, a single recessive factor, a sex-linked recessive and dominant autosomal gene, two recessive genes (one sex-linked) or a dominant recessive or a recessive autosomal gene meets with difficulties in application. The conclusion is reached that the simple formula that most nearly approaches agreement with the findings is that of two dominant genes, of which one is sex-linked. No geneticist believes that the genes which shape the construction and constitution of the body constitute the whole basis of the transmission of a morbid condition or a malformation. Characteristics are not transmitted from parent to offspring. What is inherited is hereditary units which interact and give the plant or animal the power to respond in a certain way to a given kind of environment. The genes determine the possibilities; the realization of any possibility is dependent also on environmental stimuli. The outcome is due to the interplay of environment on hereditary factors. The fact that a smaller percentage of otosclerosis occurs in some families than in others shows that influences other than the gene are at work.

DISCUSSION

DR. MELLO: In Portugal, for the last twenty years all patients with otosclerosis have learned lip reading before they became totally deaf; this has been the situation in Germany for the last thirty years, due to the work of Hartmann. The results of operations for otosclerosis performed by Passow and Barany have been poor. The patient's hearing deteriorates either immediately after operation or after a short period of amelioration following the operation. Otosclerosis is a clinical conception of progressive deafness uninfluenced by the aeration of the middle ear. If most otosclerotic patients were observed until death, the alterations so brilliantly demonstrated by Dr. Mayer would not be found in all cases. Since otosclerosis is a syndrome, no uniform therapy can be successfully administered to all patients. No progress will be made in treating otosclerosis until a clinical distinction can be made between all the types of deafness that it includes.

DR. ALAGNA: My assistant, Dr. Cusenga, in his research on experimental acidosis in rabbits, distinguished two stages of lesions in the labyrinthine capsule: (1) the resorption stage, characterized by the presence of osteoclasts, and (2) the

opposition stage of new osteoid growth, characterized by the presence of osteoblastic medullary spaces (fibroblasts, osteoblasts, etc.).

DR. MAYER: Dr. Alagna's specimens undoubtedly represent the osseous transformations of the labyrinthine capsule of the rabbit. Not every transformation is otosclerosis. Otosclerosis is characterized by primary plexiform bone tissue. It is difficult to judge animal experiments in the study of otosclerosis because every part of the human labyrinthine capsule is known while every part of the animal labyrinthine capsule is not known. Treatment is difficult because otosclerosis is a natural process of healing against which it is both difficult and without purpose to fight. A temporary improvement might be attained by making an opening in the labyrinthine capsule as Barany, Holmgren and others have done. The natural reparation of the bone, however, is opposed to the completion of the operation. It is yet to be seen whether it is possible to keep an opening in the bone of the capsule permanently open.

TYMPANIC PRESCLEROSIS OF CITELLI. DR. PAOLO CARCÓ, Catania, Sicily.

THE BLUE MANTLES IN OTOSCLEROSIS: A CONTRIBUTION TO THE PATHOLOGY OF THE LABYRINTHINE CAPSULE. DR. MORITZ WEBER, San Francisco.

EDUCATION OF PATIENTS WITH OTOSCLEROSIS. DR. G. DE PARREL, Paris, France.

OTOSCLEROSIS AND SYPHILIS. DR. FAVRE FIRMIN, Toulouse, France.

RESULTS OF FIVE YEARS OF RESEARCH ON THE SURGICAL TREATMENT OF OTOSCLEROSIS. DR. M. SOURDILLE, Nantes, France.

A NEW METHOD OF TREATING SCLEROTIC DISEASES OF THE EAR. DR. J. CAMPOS, Madrid, Spain.

IS THERE AN ELECTROCHEMICAL FACTOR IN THE PATHOGENESIS OF OTOSCLEROSIS. DR. F. LEIRI, Helsingfors, Finland.

TWO CASES OF OTOSCLEROSIS. DR. R. VILAR SANCHO and DR. ERNESTO ALONZO, Madrid, Spain.

ETIOLOGY, PATHOLOGY AND BACTERIOLOGY OF OZENA. DR. LEROUX-ROBERT, Paris, France, and DR. COSTINIU, Bucharest, Rumania.

Ozena is an acquired disease, not exceptional in new-born infants and in small children. It occurs commonly between the ages of 10 and 20 years, and rarely after the age of 45. There are three females to every male among patients with the disease. The highest incidence is in the yellow race, and the next highest in the white race; Negroes are immune on the African continent but not in other parts of the world. The condition is found mainly in persons of the lower classes among whom both living and working conditions are poor. There is a hereditary predisposition to ozena in many of these persons.

Atrophic ozenous rhinitis is due to two distinct elements, atrophy and infection. The atrophy results from neuritis of the trigeminus nerve by a descending process (lesion of the sphenopalatine block) or by an ascending process striking the nerve endings (ethmoidal ramifications of the nasal branch of the ophthalmic nerve and sphenopalatine nerve of the maxilla). These neuritides are produced by toxins of a general disease (syphilis or tuberculosis) or of a local disease (rhinitis). Their frequent appearance during the age of puberty shows that they are in relation to an endocrine dysfunction. The toxins are generally situated close to the pituitary gland because they are "rhinotropes" and because the pituitary gland represents an emunctory way of economy. The nerve endings plunge into these two types of toxins. This leads to an ascending neuritis produced by intoxication. Moreover, it can lead to a more direct endocrinosympathetic action. An atrophic stage due to paralysis of the nerve endings follows a hypertrophic stage brought

on by irritations of the nerve endings or by local hyperintoxication. A disturbance in calcium metabolism due to endocrine dysfunction favors osseous atrophy. The atrophy causes defective ventilation and furnishes a favorable culture medium. It forms the basis for ozena. The infection, brought on by a microbial process, causes the odor which is always a principal symptom of the disease. Whether primary or secondary, the infection is essential to ozena and is probably due to a rhinophilic microbe the characteristic odor of which is reproduced on culture.

Examination of the nasal chambers after common infections in childhood, especially during the age of puberty, helps to prevent the disease. The infection is treated with autovaccine, monovaccine, polymicrobial vaccine or bacteriophagic vaccine after suppression of antigens. The atrophy may be treated with paraffin, with barium oxide petrolatum, not through a general stricture of the nasal chamber but by the injection of an extract of pilocarpine, acetylcholine and similar preparations, or by periarterial sympathectomy which produces swellings and humidification of the mucosa.

SURGICAL TREATMENT OF OZENA. DR. A. LAUTENSCHLÄGER, Berlin, Germany.

Ozena is an inflammatory process of the mucosa of the nose and its adjacent cavities. The symptoms are atrophy, formation of crusts and fetor. Rhinitis atrophicans is characterized by connective tissue-like degeneration of the mucosa, primarily of the periosteal layer. Decomposition of the nasal conchas, shriveling of the cavernous plexus of the mucosa of the inferior concha, loss of specific mucous elements and disturbances of the fluid in the interior of the nose result therefrom. The accessory sinuses are involved as a result of the inflammatory process; the osseous walls of the accessory cavities are thickened. With the disturbance of specific mucous elements through the connective tissue degeneration, the natural moistening of the nasal mucosa decreases and the pavement epithelium increases. In the nasal cavity, widened by the shrinking of the mucosa and the decomposition of the nasal conchas, the pushed-off pavement epithelium mixes with the inflammatory products of the mucosa and the thickened secretion dries on the fluid-deficient surface of the mucosa under the influence of the air coming through the nasal cavity.

Operative therapy aims to restore as much as possible the function of the nasal mucosa. This objective is reached: (1) by the removal of the inflammation causing pathologic differences, (2) by retaining specific elements of the mucosa and increasing their function (operative prophylaxis) and (3) by compensation of the fluid exchange in the internal part of the nose.

Every operation on the diseased mucosa causes a stronger perfusion of blood in the mucosa and bone and therewith an increase of the mucous function and an improvement of the fluid exchange. In order to produce lasting effects the operation should be performed on the side wall of the nose. Through considerable dislocation of the nasal wall and through multiple infractions of the bone, regeneration centers are created in which new growths occur. From then on the zones of mucosa still capable of function are better nourished, there is better perfusion of blood in the mucous membrane, and the hyaline connective tissue is reabsorbed (operative reparation).

The compensation of fluid exchange is assured in the following way: (1) The increased blood supply resulting from the operation restores the stream of fluid which permanently irrigates the surface of the mucosa and originates directly from the vascular system; (2) sufficient operative contraction of the nasal sinuses hinders the excessive delivery of fluid to the respiratory air, and (3) the remaining lack of fluid balance is compensated by the parenteral administration of physiologically equivalent fluid (salt solution or Ringer's solution) or by the implantation of the excretory duct of the parotid gland in the antrum of Highmore. The earlier the operative therapy, the easier the individual tasks may be performed and the better the results.

MEDICAL TREATMENT OF OZENA. DR. VIGGO SCHMIDT, Copenhagen, Denmark.

The initial stage of ozena generally occurs during childhood and early puberty. It is frequently too late to check the condition when the infectious process has continued for so many years that it presents characteristic symptoms. Clinical evidence shows that ozena is often familial; thus many of these patients may be detected during their early childhood by a study of their family trees. A new-born infant in an ozenous family must be given special general and hygienic treatment, with appropriate nutrition, and infection of the child by the mother or other ozenous patients must be guarded against by the rhinologic method (vaccination of the nose with encapsulated bacteria). In the event that a child is found to have ozena in an early stage he should be given the usual dietetic and hygienic treatment together with the vaccine; whether or not a child shows a tendency to the disease, the treatment can do no harm.

Vaccine therapy is applied when the processes are chronic; protein therapy, when they are acute and febrile. Both treatments can be used only when there are no isolated infection cavities (such as abscess cavities or sequestrums). The general condition of a patient should be improved before medical treatment is concentrated on the diseased organ; thus insulin has been used in the treatment of ozena in order to stimulate, not merely pancreatic function, but the entire system, thereby offering indirect temporary aid in the combating of the ozena. Treatment with preparations of thymus, pituitary, thyroid and ovary temporarily affect the vegetative nervous system as well as the nasal mucosa.

Acriflavine hydrochloride, powder of Quillaia bark, swabbing with pilocarpine and diathermy have given excellent results in the local treatment. Avitaminosis is often associated with this condition. Some authors claim that a lack of vitamins A and D during fetal life and early childhood is a frequent cause. They propose vitamin treatment during the latter period in order to make up for the deficiency and to increase resistance to infection. There is no reliable form of medical treatment of ozena. No remedy can be successfully administered to even a large percentage of patients. The best method appears to be vaccination with an autogenous vaccine of encapsulated diplococci. Combined surgical and medical treatment has given the best results.

PATHOLOGIC ANATOMY OF OZENA. DR. U. L. TORRINI, Florence, Italy.

Many patients with ozena have flat noses, slightly saddle back in type, with raised and dilated nostrils. A tilting of the nose is due perhaps to the respiratory strain at the beginning of the disease and later, in the atrophic stage, to the presence of crusts which hinder normal circulation of air. The initial hypertrophy of ozena has no characteristics that distinguish it from common subacute purulent rhinitis. The lower and middle turbinate bones appear hypertrophied, and the mucosa is sprinkled with a nonodoriferous, gray, puslike fluid. The secretion thickens mostly in the middle meatus, which implies that the anterior and middle ethmoid cells participate in the suppurative process. The lesions are bilateral and vary in intensity; if one of the nasal fossae is stenosed by a spur or crest of the septum, it may be undamaged while the other may be in an advanced stage of atrophy. Since the tumid mucosa secretes during a marked reduction of the inferior turbinate, it is felt that the atrophic process begins in the bone tissue and thence spreads to the mucosa. If this were not so, it could not be explained how, with an atrophy so accentuated, the mucosa could keep its epithelium macroscopically intact and the glandular function sufficiently active, as the humidity of its surface demonstrates.

Various bacteria are found in the nasal pus and mucopus of patients with ozena. Some of these micro-organisms are considered agents of putrefaction or fermentolytics and are thought to cause the fetor. According to some authors, ozena begins in the sinus cavities and may be cured only by treatment of the sinuses. There are more dangers than benefits in this therapy. The paranasal sinuses are generally undamaged, and if lesions are present in them, they are

secondary. Ozenous lesions are not limited to the nasal cavity but constantly spread to the nasopharyngeal and to the pharyngobuccal cavities.

In an advanced state of ozena the mucosa becomes dry as a result of insufficient glandular secretion. The pus hardens into crusts, and the mucosa soon becomes opaque and glassy through the cornification of the superficial layers of the epithelium. The ozenous lesions do not occur exclusively in the nasal fossa but may appear on all the mucosa of the respiratory tract as a propagation of the nasal lesion or more rarely of the primary alteration. Since the most habitual seat of ozenous lesions is the nose and nasopharynx, involvement of the ear may occur through the eustachian tube and involvement of the eye through the nasolacrimal canal.

The cylindric epithelium is transformed into pavement epithelium. The pluristratified epithelium varies in thickness at various points in the section. The cells constituting the epithelial layer are generally irregular in form and size and often have rather irregular nuclei, frequently impelled to the periphery. In the epithelium is a strong infiltration of parvicellular elements arising from the substratum in which there is a thickening of these elements. In the infiltration elements there is a scarcity of plasma cells. The basal layers of the epithelium often reveal an abundant series of cylindric, almost cubic, cells. This metaplastic process is thought by many to be the cause of the characteristic fetor of ozena. The less intense the parvicellular infiltration, the less secretion stagnates in the nasal cavity. The epithelial substratum normally consists of a fibrillary connective tissue with few elastic fibers; in ozena there is thickening of these connective fibers through the reduction and disappearance of the interfibrillary spaces. The thickening and condensing of the connective fibers into bundles are partially the result of (1) a connective proliferation which develops within the blood vessels and glands, (2) the resorption and disappearance of the mucus with which the mucosa is normally imbued, and (3) the retraction of the blood vessels together with atrophy of the numerous glandular tubes. As a result of these factors fibrosclerotic tissue develops. When the atrophy is completely advanced and the mucosa completely sclerosed, the thickening of the elastic fibers is rare and sometimes disappears.

It is evident that the tissues undergo an atrophic-sclerotic process: metaplasia and atrophy of the epithelium; atrophy of the glands; atrophy of the osseous tissue, and sclerosis of the epithelial substratum (tunic) and of the blood vessels. This atrophic-sclerotic process is never complete even in the more advanced stages. Thus the epithelial metaplasia evinces here and there groups of cells which are always of the cylindric or cubic type. The atrophy of the glands is never complete. Accurate biopsies reveal the presence either of glandular openings in the epithelial stratum or of rare tubules in the tunic with profoundly altered cellular elements. The proof that there is never complete transformation or atrophy of these elements lies in the fact that they almost reacquire their primary characteristics and reappear when the tissues are treated.

OZENA: BACTERIOLOGY AND EXPERIMENTAL PATHOLOGY. DR. A. TRIMARCHI, Cremona, Italy.

Whether ozena is a constitutional or a bacterial disease has been a matter of controversy. Perez, Hofer and others recognize it as an infectious disease and believe that they have demonstrated the possibility of cure following vaccine therapy.

Ozenous micro-organisms are divided into three groups: encapsulated bacteria, pseudodiphtheria bacteria and bacilli of Perez. Löwenberg described for the first time, in 1881, a micro-organism obtained in pure culture from the nasal mucus of patients with ozena. It was presented as a large, encapsulated coccus, nonmotile, occurring in pairs and similar to Bacterium mucosum-capsulatum. The characteristic fetor was not reproduced on culture.

In 1893, Abel stated that in the secretion between the crusts and mucosa there existed a large, encapsulated bacillus, occurring in pairs and never absent in

cases of ozena. It was 1.25 microns in length and developed abundantly on plates of agar and gelatin, together with other bacterial forms. A gelatin culture showed vigorous development on the surface; infixion was very poor. In agar, blood serum, potato and milk (at 37 C.) the development took place in twenty-four hours. The bacillus stained well with aniline dye, and the capsules were rendered apparent with methylene blue (methylthionine chloride, U. S. P.) and acid fuchsin.

The similarity of this organism to Friedländer's bacillus and its identity with the bacillus described by Löwenberg gave it the name Löwenberg-Abel bacillus. Many authors believe that it is of great importance in the production of ozena. It is found in a majority of cases. Some authors have stated that it can be found in the nasal mucus and the frontal and maxillary sinuses of healthy persons. According to Anthon, it is always present in pharyngeal keratoses.

Differentiation of encapsulated bacilli is rendered doubtful by the uncertainties of agglutination reactions. The results of vaccine therapy with these organisms have not been noteworthy.

Diphtheria bacilli and similar organisms are gram-positive. They are found both in the crusts of ozena and in the depths of the tissues. The isolation and cultivation of these micro-organisms are not always possible because they are strongly hindered by the vigorous development of the Löwenberg-Abel bacillus. This micro-organism is similar to the diphtheria bacillus but differs from that of pseudodiphtheria by its slight production of toxin and its scanty development of agar. Della Vedova distinguished between chronic ozenous rhinitis of rapid evolution with atrophy and intense fetor, attributed to the diphtheria bacillus, and chronic rhinitis of slow evolution and slow secretion of fluid, due to the Löwenberg-Abel bacillus. Most authors agree that the results of serotherapy, namely, fluidification of secretion, attenuation of bacteria and disappearance of fetor, are temporary and that there is a disproportion between the therapeutic and the curative effect.

These organisms have no specificity according to the results of most research. Absolute proof has not been obtained that the various forms of pseudodiphtheria bacilli are atypical diphtheria bacilli, nor have means been found to classify them definitely.

Perez's bacillus, called Coccobacillus foetidus-ozaenae, is gram-negative but takes all aniline stains; it is polymorphic and varies in dimensions and form. Its culture reproduces the fetor. It is found in both the saliva and the nasal secretion. Hofer and others maintain that vaccinotherapy with Perez's bacillus is the only means of securing a permanent cure of ozena. Injections of Perez's vaccine cause nasal reactions, manifested by severe nosebleeds. The organism is easily demonstrated on culture. Some consider the Perez bacillus a member of the genus Proteus while others identify it with the Pasteurella group.

Other micro-organisms found in the crusts and secretion of ozena are: Bacillus of Pez and Gradenigo, Bacillus coli, Bacillus proteus, Bacterium oxygenes, Bacillus fluorescens, Bacillus pyocyaneus, Bacillus putidus, Streptococcus, Pneumococcus, Bacillus influenzae, Micrococcus catarrhalis, Staphylococcus aureus, and acid-resisting bacillus, spirilla, anaerobes and filtrable organisms.

Abel was the first to produce ozena by introducing a culture of Löwenberg-Abel bacilli into the nasal cavity of a patient with advanced tuberculosis. He was unsuccesstul, however, in reproducing the disease in animals. According to Perez, the injection into the marginal vein of a rabbit of from 0.5 to 1 cc. of a culture of C. foetidus-ozaenae produces necrosis of the auricular cartilage and hyperemia of the pituitary gland after twenty-four hours. Greater doses kill the animal. Repeated injections of small doses produce chronic rhinitis with atrophy of the inferior turbinate. Many authors have met with negative results in experimenting with one of these two organisms.

Wirth stated that in the nasal secretion of rabbits there are gram-negative mobile elements which illustrate all forms from typical B. fluorescens to the bacillus of Perez. It is rather difficult to differentiate between these micro-organisms

resembling Perez's bacillus solely on the basis of cultures. According to Wirth, one must resort to agglutination reactions.

Experiments have demonstrated that the bacteria by themselves are not capable of producing true and permanent ozena, nor can they grow in or even penetrate into the deep layers of the epithelium in the absence of peculiar circumstances. That is why de Simoni considered ozena as a very complex lesion into which an anatomic factor and one or more organisms must necessarily enter.

DISCUSSION

DR. CANESTRO: The fundamental factor in ozena is the local constitution and the recognition of the constitutional status. The local constitution is determined not only by congenital and hereditary causes (special functional defects of the glands directly in correlation with the development in the nose and its mucosa, such as the thyroid, pituitary and genital glands) and by neurotrophic or general trophic causes, such as avitaminosis.

DR. LEIRI: Like the blood and blood serum, the nasal secretion in ozena has a dielectric constant similar to that of water. The double layer potential between the nasal secretion and the mucosa develops according to Coehn's rule, namely, that the nasal wall acquires a negative charge while the secretion acquires a positive one. When in ozena the secretion dries in a crust, the nasal wall is covered with porous membranes through which the water in the secretion oozes toward the surface. As a result of the movement of the water through the pores there arises a current potential, in that the negative charge accumulates at the mucosa while the positive charge moves with the water to the surface of the crusts where it probably is torn away along with the air current. Thus a greater negative charge appears normal in the nasal mucosa. This negative charge attracts the positively charged calcium ions in the bone fluid, especially in the turbinated bones where there is a negative charge on both sides of the bone. As a consequence of this attempt toward the diminution of calcium concentration, new ions go into solution, thereby resulting in dissolution and absorption of bone.

This also explains the dissolution and absorption of bone in otitis media with cholesteatoma.

DR. BARUCHOFF: On the basis of examination of thirty-four patients it was concluded that development of ozena is not influenced by age or nationality. These patients included Bulgarians, Jews and persons of other nationalities. Brief reports of four of the most interesting cases follow: 1. A boy, aged 9, suffering from unilateral ozena, was treated by the usual means, and after puberty the ozena improved. The fetor and crusts almost disappeared. 2 and 3. Two women, aged 46 and 52, acquired ozena immediately after the beginning of the symptoms of the menopause. When the symptoms were treated the fetor and crusts were relieved. 4. In a girl a goiter developed simultaneously with ozena. The possible relation between ozena and the endocrine glands should be investigated.

DR. MOTTA: One may conclude that the mycelia found in the external ambient penetrate early into the nose. Deficiency in the respiratory current leads to clogging of the nose, especially if hygienic conditions are neglected, and the mycelia find in the purulent and crusty nasal secretion the food adapted to their saprophytic life.

DR. LASKIEWSKI: The roentgenograms in fifty cases of ozena in which the patients were treated in the clinic at Pozen showed three types of cases: Type 1 includes cases in which there is normal development of the accessory sinuses; the contours of the bony ridge which surrounds the auxiliary cavities as well as the contours of the ridge of the nasal septum are thickened. Type 2 includes cases in which one frontal sinus is not seen in the roentgenogram and in the region of the ethmoid sinus there is a light spot which sometimes is so large that it appears like a pneumatic space. Type 3 includes rare cases in which one frontal sinus is not seen in the roentgenogram and the contours of the ethmoid sinus and of both maxillary sinuses are greatly narrowed. One finds considerable thickening of the

outer bony wall of the maxillary sinuses and of the orbital ridge of the contours of the zygoma and the bony ridge of the nasal septum. Microscopic examination of specimens in cases of type 3 revealed a compact structure of the bone and atrophy of the blood vessels; the arterial blood vessels of the periosteal layer showed considerable thickening of all layers. The roentgen shadow of the ethmoid sinus and especially that of the maxillary sinus depend in no way on a disease of the mucosa of the involved accessory sinuses but on the enormous condensation of the facial accessory sinus wall. The contours of the sella turcica in patient with manifestations of type 3 were narrowed but were otherwise normal. The earlier the atrophic process develops in the interior of the nose, the less roentgen shadow of the accessory sinuses there will be.

DR. BILANCIONI: The familial aspect of ozena should be stressed and investigated accordingly. Not all the attention should be given to the ozenous patient, but his family should be investigated as well.

DR. MELLO: In order to get a clear idea of the value of various types of treatment, it is important to observe patients regularly for a number of years after they are discharged from the clinics and hospitals. Twenty-three patients with ozena were given rhinoscopic treatment once a year for eleven years. The patients were mainly females, aged from 8 to 23 years. The parents of many of them suffered from alcoholism. The patients were all given general treatment, including heliotherapy and cod liver oil. Local bathing was done with a light hypertonic salt solution. The crusts and fetor soon disappeared. The mucosa appeared almost normal. After four years three of the patients gave unmistakable signs of hypertrophic rhinitis. This number is too small, however, to justify any general conclusions.

If the patient comes to the physician fairly early and ordinary treatment proves of no avail, the specialist should resort to surgical therapy.

THE SIGN OF BILANCIONI IN OZENOUS ATROPHIC RHINITIS. DR. FEDERICO BRUNETTI, Venice, Italy.

TREATMENT OF OZENA BY A COMBINATION OF BACTERIOPHAGE AND PHARMACOLOGIC VASODILATATION. DR. DJIROPOULOS and DR. HALPHEN, Paris, France.

A PERSONAL METHOD OF INTRANASAL OPERATION FOR OZENA: RESULTS OBTAINABLE. DR. HALLE, Berlin, Germany.

HISTOLOGIC AND BACTERIOLOGIC RESEARCH ON OZENA. DR. HENRI PROBY, Lyons, France.

PARAFFINOPLASTIC THERAPY FOR OZENA IS NOT ONLY CLINICAL BUT ANATOMICOPATHOLOGIC. DR. U. L. TORRINI, Florence, Italy.

SURGICAL TREATMENT OF OZENA BY SUBPERICHONDRAL CARTILAGINOUS GRAFT. DR. M. BALLERIN, Paris, France.

SURGICAL TREATMENT OF OZENA. DR. BLAU, Gorlik, Poland.

THE ANATOMICOPATHOLOGIC AND THERAPEUTIC STUDY OF OZENA. DR. GUNS, Paris, and DR. PICARD, Louvain, Belgium.

DISCUSSION

DR. BLAU: Dr. Halphen stated that it is impossible for an atrophic turbinated bone to reassume its normal form, color and general characteristics. According to the experience of most physicians, that is totally incorrect. On the contrary, exceptionally severe hypertrophies may appear. The entire question depends on whether many mucous elements are contained in the distant nasal cavities. Most significant is the lymph stream resulting from the strong perfusion of blood after the numerous injuries to the bone during operation and the development of vascular neoplasms.

DR. SZMURLO: Ozena-like rhinoscleroma is found most often in young girls. In the treatment of ozena excellent results were obtained by means of the inhalation of hot hypertonic solutions of from 3 to 5 per cent sodium chloride for from twenty to thirty minutes a day. There were also good results from the use of tampons saturated in a warm 10 per cent aqueous solution of mild silver protein which were introduced into the nasal interior toward the middle meatus. In the clinic in Vilna autohemotherapy was successfully administered in rare cases.

DR. HALLE: Patients should be given all types of treatment for ozena. No outstanding success has been obtained with any particular method. My own method has proved useful. It is true that an atrophied turbinate bone can acquire a normal appearance again. The mucosa becomes damp and red and looks normal. In many cases the sense of smell, which may have disappeared, returns.

DR. MELLO: Dr. Halphen affirmed that an atrophied turbinated bone cannot be regenerated. This is in striking contradiction to the experience of most physicians. After cure there may be even hypertrophy of the mucosa.

DR. ANTOLI CANDELA: I have never cured an ozenous patient with treatment exclusively by vaccination or any other form of therapy. Vaccination accompanied by repeated irrigation with isotonic solutions and swabbing of the mucosa has aided in curing a number of patients who were given constant treatment for many years. Surgical treatment is doubtful, uncertain in its results and not to be advised in general.

THE HISTORY OF SCLEROMA. DR. M. HAJEK, Vienna, Austria.

Dr. Hajek discussed the changes in the concept of rhinoscleroma, beginning with Hebra's first publication in 1870. A study of the clinical picture soon shows that the disease may appear in the pharynx and glottis as well as in the nose. Most cases of subglottic laryngitis and of Stoerk's blennorrhea may be considered as forms of scleroma.

The disease is frequently found in the eastern European countries, where it constantly tends to increase. The etiology of scleroma is more disputed than the clinical picture. Despite meritorious investigation, there is nothing but probable evidence of the etiologic significance of the scleroma bacillus. While the finding of the bacillus of Frisch in Mikulicz's cells is of great etiologic importance, the complete similarity of this organism in culture to the bacillus of Friedländer and Abel contraindicates the specific character of the scleroma bacillus.

The extensive bacteriologic research in which differentiation of the encapsulated bacilli was attempted on the basis of their different reactions to sugar solutions cannot be considered as specific evidence.

The results of inoculation of animals are likewise unconvincing, even though Stepanow, Kraus and Šercer succeeded in producing at the site of vaccination in white mice infiltrations the histologic structure of which resembled that of scleroma tissue. Inoculation of human beings has given completely negative results. The serologic reactions of Goldzieher and Neuber (complement-binding allergy) furnish definite evidence of the specificity of the scleroma bacillus, provided that the results coincide with those obtained on reexamination.

The pathogenesis of scleroma is vague. Nothing is known of the manner of infection or as to whether the disease is contagious. Thus an important factor in fighting the disease is lacking.

ETIOLOGY OF SCLEROMA. DR. HERMANN STREIT, Königsberg, Germany.

Of the serologic methods for the differential diagnosis of scleroma the test for deviation of the complement (Bordet-Gengou) has offered the best results. The agglutination method is often considered equal to the test for deviation of the complement. The agglutination method, as compared with the complement-fixation test, is more practicable and simple and hence preferred. The primary seat of scleroma is usually in the nose. Primary laryngeal scleroma is rare. The A parvicellular infiltration is often found under the epithelial cornea, consisting and give rise to connective tissue. Mikulicz's cells are also large and of diverse

author presumes that the bacteria usually penetrate the mucosal epithelium directly; they may often be eliminated by prophylactic measures, or at least hindered from further development. The typical cellular degeneration of scleroma is a result of the specific bacterial influence.

Generally, scleroma bacilli are found in the lymph spaces or the tissue cells. Sometimes no bacteria are found in sclerotic tissue because of faulty staining technic or hardening of the tissue. Sometimes the Mikulicz cells are already evident in the clinical atrophic catarrhal stage of scleroma. Attempts to produce scleroma experimentally have been predominatingly unsuccessful. Histologically, the experimentally produced scleroma bacilli differ from the Friedländer bacilli in human beings.

Scleroma progresses rapidly and shows a great tendency to spread. One of the greatest endemic seats of the disease is Poland, whence it spreads westward. Most patients with scleroma are farmers. More emphasis has been placed lately on the study of the familial transmission of the disease.

In man, all attempts at inoculation—namely, the injection of scleromatous liquid into the cheeks and ears (Rona), the insertion of tampons saturated with scleromatous liquid into the nasal cavities (de Simoni) and the implantation of a piece of scleromatous tissue into the tegument of the arm (autoinoculation of Schrötter)—have been unsuccessful. Except for the bacillus of Frisch, the results are not specific. Nothing is known of the manner of transmission of the disease to man. It is said that the disease is not directly transmittable from person to person, but that it may be transmitted to man only under certain conditions by fleas, lice and common scab-nits.

Scleroma is often found in certain conditions of personal uncleanliness, nasal obstruction, poor aeration or lack of hygiene, especially in countries where close contacts exist between scleromatous patients and healthy persons. The disease, according to some authors, is not of alimentary origin and is not hereditary. According to the scientific knowledge of today all that can be said is: (1) Scleroma is an infectious disease; (2) the causative agent is with great probability the bacillus of Frisch, and (3) the characteristic alterations of the tissues are due to the action of this bacillus.

ANATOMICOPATHOLOGY OF SCLEROMA. DR. JEAN SZMURLO, Wilno, Poland.

Scleroma is primarily localized in the upper respiratory tract. Secondarily it can appear at the vestibule of the nose about the pyriform aperture, on the upper lip and along the nasopharynx. Pavlov observed scleromatous infiltration of the arm (after inoculation) and spreading of the scleromatous lesions from the interior of the nose to the lacrimal passages and the eustachian tube, whence they penetrated the tympanic cavity and even appeared in the external auditory meatus. Rare sites of secondary localizations are the tongue, the lower buccal cavity and the ocular orbit. In the respiratory tract scleroma is most often localized in the interior of the nose, in the vestibule or in the posterior nares. In the larynx the scleromatous infiltrations are found usually in the subglottal space; they are seen sometimes on the ventricular strips and more rarely on the aryepiglottic folds and on the posterior surface of the epiglottis.

The pharynx and throat are the seat of the lesions in a third of the cases; the lesions are found in the mesopharynx and occupy the posterior surface of the soft palate, the uvula, the pillars and the lateral parts of the posterior wall. Though rarely the seat of scleroma, the trachea sometimes has lesions in its upper part near the larynx. They are sometimes observed in the inferior part, close to the bifurcation, whence they descend rapidly to the bronchi.

The anatomic lesions of scleroma are either diffuse or circumscribed. In some cases, the lesions are diffuse in one organ and circumscribed in another organ. The diffuse form first appears as atrophic rhinitis characterized by atrophy of the nasal mucosa with abundant secretion and gray crusts on the surface. The atrophic lesions extend along the pharyngeal mucosa, descending to the larynx

and trachea, where the characteristic crusts are formed. Besides the atrophic rhinitis there is a diffuse infiltration of the mucosa which extends to the neighboring regions and occupies the subcutaneous tissue, passing to the vestibule of the nose, to the tissue around the pyriform aperture and to the nasopharynx, the lips and the gums. Diffuse infiltration also strikes the pharynx, extending to the mucosa of the soft palate, the uvula and the pillars. It is found in the subglottal part of the larynx after causing stenosis with ensuing attacks of suffocation.

In the nasal vestibule the diffuse infiltration spreads to the septum, to the floor of the cavity and to the lateral wall, closing the nostrils considerably. In general, there is a symmetry of scleromatous lesions. Diffuse infiltration sometimes attacks the lateral wall of the laryngeal vestibule, making it rigid and hindering its movements and those of the subglottic region. The infiltrated mucosa becomes thickened, stiff and of a pale rose color with deep stains where the epithelium is eroded. The infiltrations of subcutaneous tissue, after having become too large, adhere to the skin of the lip or the cheek and, by compressing it, cause atrophy.

The circumscribed infiltrations appear on the entire surface of the upper respiratory tract. They occur in nodosities of different types and sizes. There are small nodules the size of grains of millet or hemp or small peas and large nodules the size of French beans or cherry, almond or prune pits. The small nodules habitually appear in groups, but they have been observed singly on the posterior wall of the epiglottis close to its free edge on the aryepiglottic fold and on the posterior wall of the pharynx. The large nodules develop in the septum of the nose, the ear trumpets and the posterior wall of the epipharynx; in the trachea they obstruct the passage of air. They are of rose color and are distinguished from the mucosa by a deeper tint. They are flat, somewhat spherical or conical. Their consistency varies according to age; the early infiltrates are soft, while the older ones become hard. The small nodules become enlarged, unite little by little and form a confluent group. The diffuse or circumscribed lesions which occur on the internal and external walls of the nasal vestibule, the two walls of the subglottic space, the posterior surface of the posterior pillar or of the soft palate and the posterior wall of the pharynx, by enlarging, gradually lessen the space that separates them.

Scleroma is an infectious granulomatosis, produced by the encapsulated diplobacillus and its toxins. Scleromatous lesions develop habitually on the mucosa of the upper respiratory tract, never in the cartilage and bones. During the first stage the epithelium is transformed from ciliated to stratified pavement cells. In some deeper cells of the cornea the protoplasm becomes more dispersed and partially disappears, vacuoles appear and the nuclei become less voluminous and pyknotic. A parvicellular infiltration is often found under the epithelial cornea, consisting of small and large lymphocytes, polymorphonuclears, plasma cells and sometimes very large protoplasmic cells in the form of a flimsy network, most often situated close to the periphery. These protoplasmic cells are Mikulicz's cells. Homogeneous globules of different sizes are found, which stain deeply with acid fuchsin. These are Russell's bodies. Besides the Russell bodies there are homogeneous masses in places which stain well with eosin and acid fuchsin. The vessels are numerous and sometimes dilated, and in many there is an increased number of endothelial cells. The length of the vessels of the bundles of connective tissue which penetrate the interior of the cellular conglomeration is next observed. The plasma cells undergo various modifications. The protoplasm gradually melts and disappears, the nuclei become free and undergo karyokinesis, and the destruction of the cells gradually ends. The cells then begin to grow, become homogeneous and undergo hyaline degeneration. They are even transformed into fibroblasts and give rise to connective tissue. Mikulicz's cells are also large and of diverse forms. Large cells with voluminous vacuoles, filiform protoplasm and small pyknotic nuclei are found; some are the size of plasma cells, containing much protoplasm, small vacuoles and one or two nuclei of ordinary volume; some contain diplobacilli which are colored distinctly by hematoxylin or by the Gram stain as modified by Weigert. The diminished parvicellular infiltration is gradually replaced by fibrous connective tissue which hinders the formation of necrotic foci

despite a live process of disintegration. The plasma cells and Mikulicz's cells disappear, and only the lymphocytes and rare Russell's bodies are seen. The development of collagenic fibrous connective tissue is most accentuated around the vessels. In some cases there are considerable increase and multiplication of the endothelium; some vessels are thrombosed, and their walls present hyalinization. The same degenerative process may be observed in the connective tissue which is transformed into cicatricial tissue. The hyalinization is attributed to the activity of the bacillus of Frisch.

Scleroma is a local disease; its generalization has never been observed in man as in mice. A growth in the size of the cervical glands is occasionally produced by secondary infection. Lesions of the heart and lungs are caused by an obstruction in the respiratory tract, especially in the larynx and trachea, by scleromatous infiltrations which cause a diminution of the intake of air in the lungs, a lowering of intrathoracic pressure, an obstruction of the pulmonary vessels or atelectasis of the alveolus. Hypertrophy of the right side of the heart occurs, and in turn fatty degeneration of the cardiac muscle. This fatty degeneration is in the majority of cases the immediate cause of the death of scleromatous patients. Death sometimes occurs after operations for the removal of the scleromatous masses obstructing the larynx and trachea. Sometimes fatal mediastinitis, purulent pleurisy or even adhesive pericarditis develops. Sometimes death is caused by suffocation as a result of the scleromatous masses in the trachea. Some authors feel that there is absolute incompatibility between scleroma and tuberculosis, whereas others maintain that since scleroma causes a catarrhal state of the upper respiratory passages it favors infection by the tubercle bacillus. There are cases in which scleroma is complicated by syphilis and neoplasms.

EXPERIMENTAL PATHOLOGY OF SCLERODERMA. PROF. A. ŠERCER, Zagreb, Jugoslavia.

Scleroma undoubtedly belongs to the group of infectious granulomas which under the effect of a specific virus form a specific granulation tissue. Scleroma is characterized by (1) a specific virus, (2) a specific histologic substratum and (3) a specific clinical picture. Research on scleroma began immediately after the discovery of the scleroma bacillus by Frisch and Pelizzari in 1882. The first experiments were, however, completely unsuccessful.

With the assumption that a longer time was necessary for the development of scleroma, most authors gave little consideration to the phenomena occurring immediately after inoculation.

Kraus discovered in 1907 that a long time was not necessary for the development of a typical scleromatous infiltrate, but that in white mice the infiltrate showed all the characteristic indurations of fresh scleromatous foci in human beings in from eight to ten days.

Only in the most recent serologic and bacteriologic investigations have the experimenters succeeded in distinguishing scleroma bacilli from other encapsulated bacteria. As a result of this, the new inoculation experiments, which were made with specific scleroma bacteria, must be afforded a specific value. In 1924 Kraus renewed his experiments and was able to substantiate the results obtained in 1907.

Nyfield obtained the same favorable results in 1925 with twenty-five guinea-pigs, and Šercer in 927 with fifty white mice. By histologic examination they were able to determine the filtrates which offered a lively proliferation of connective tissue, with leukocytes, plasma cells, Russell's bodies and genuine Mikulicz cells.

The fact that the authors mentioned could not produce granulomas with specific scleromatous elements in the mucosa of the respiratory tract but produced them in the skin, and that Stepanow by inoculation of the outer chamber of the eye likewise produced granulomas which possessed all the characteristics of human scleroma, speaks in favor of the specificity of these infiltrates. This conviction is supported by the observation of Kraus and Šercer, that after the subcutaneous injection of scleroma bacilli specific alterations are seen in the respiratory tract.

The results reported do not suffice to characterize the clinical picture of scleroma. Chronicity and the intermittent alternating of fresh and cicatrized infiltrates are missing.

In 1930 Šercer performed inoculation experiments on five apes with pure cultures of scleroma bacteria. In these animals, as in other experimental animals, temporary infiltrates were soon produced which did not lead to a clinical picture of the disease.

According to Šercer, the reason why the scleroma bacilli find favorable conditions of transmission in human beings and not in animals lies in the reciprocal effect of the bacteriophage on the bacterial capsule and of the body of the bacillus on the animal organism.

Significance of War in the Spread of Scleroma of the Upper Respiratory Passages. Dr. A. Putschkowsky, Kiev, Russia.

War has contributed to a large degree to the spread of rhinoscleroma. Even before the World War the disease was not rare; it was widely disseminated in Poland, White Russia, Ukraine, Rumania, Bulgaria, Hungary, Czecho-Slovakia, Silesia, East Prussia and other European and non-European countries. The localities now most affected are southwestern Russia, eastern Galicia, Moravia, Bohemia, Silesia, Bukowina, Rumania and southern Italy. The disease is also found in the canton of Valais in Switzerland. In the other European countries, especially in France, it is almost unknown. The last cases observed in France occurred during 1883 and 1896 in persons of foreign nationality. The wars of the last century were the real cause of the spread of scleroma, since the armies were largely recruited from Russian, Polish and Ukrainian soldiers. During the World War scleroma spread in such a way as to become a social menace to all humanity. After the war the cases of scleroma multiplied in Africa (Algeria and Morocco), in Asia (Siberia and Turkestan), in the Caucasus and in India. There is no direct evidence of scleroma in Australia, but the disease may exist there. Before the war military service was the cause of propagation. During the war the spread of the condition was brought about by the maneuvers of the troops and the displacing of the civilian population and the prisoners. Before the War the soldiers with scleroma infected others. Protective measures were taken at this time, notably in Russia, where the disease carriers were exempt from military service. During the War these precautions naturally were overlooked; the armies, together with their prisoners of war, invaded the countries where scleroma was unknown. Koenigsfeld maintains that the great increase of cases of scleroma in Prussian Silesia was caused by Russian prisoners who were concentrated there. The same may be said for Hungary. On the other hand, the Austrian and Hungarian prisoners were infected in Russia.

Scleroma is spreading rapidly and threatens to become a social danger. The various governments should lend their assistance in helping to decrease the number of people afflicted with this disease.

International Legislation on Scleroma. Dr. Burraud, Lausanne, Switzerland.

Means to prevent scleroma should be made obligatory for all countries and accomplished by international legislation. Moreover the countries should be obliged to publish statistics, as accurate as possible, showing the prevalence of the disease, the exact number of cases, the precise geographic distribution and under what particular conditions the condition occurs as well as the causes favoring it.

Bacteriology of Scleroma. D. F. Lasagna, Parma, Italy.

The bacillus of Frisch belongs to the encapsulated group, together with the bacillus of Friedländer. It is short, rounded and ciliated, with a capsule apparent

in some forms. It can be stained with orcein, acid fuchsin, hematoxylin, carmine or osmic acid, by the methods of Alvarez, de Rossi and others. It is gram-negative. After staining the bacillus is found in the tissues, in the cells of Mikulicz, in the lymphatic spaces and in the intracellular epithelial tissue; it is the cause of cellular degeneration in these locations. In agar, gelatin or potato cultures it assumes the color, disposition and typical aspects which differentiate it from other encapsulated organisms. The bacillus causes melting of the medium and the formation of acids but seldom of gases. It offers slight resistance to the action of mixed bile on the culture medium. When its cultural form is defined, it may be differentiated by agglutination from other encapsulated forms. It is present in all cases of scleroma together with other micro-organisms, in particular, encapsulated forms (Bacterium ozaenae) and the tubercle bacillus. It has a greater virulence for rats and guinea-pigs than for other animals. It leads to peritonitis, pneumonia and nephritis and causes rapid death by septicemia. It can occasion inflammatory lesions with cellular degenerations comparable to those in human beings, on intratracheal, subdural and corneal injection, especially if the inoculum is well prepared. These results, illustrated by numerous investigators, have been contraindicted by the negative results of others. The virulence of Frisch's bacillus and the inflammation caused by it do not help to distinguish it from other encapsulated bacteria. There are no encapsulated forms existing in animals which can be confused or compared to the bacillus of rhinoscleroma.

Treatment of Scleroma. Dr. Burak, Minsk, Russia.

The treatment of scleroma has seldom met with great success. Formerly, medicinal substances were often used, such as salicylates, phenol, corrosive mercuric chloride, iodine, osmic acid, alkalis, acid preparations and arsphenamine. Today, actinotherapy is most frequently applied along with roentgen radiation, which provokes complications, above all stenosis of the larynx. The quantity and quality of the rays are of great importance. A combination of roentgen radiation and electrocoagulation has met with some success. The results of diathermy vary. Authors are not all agreed on the beneficial effects of vaccinotherapy. Sometimes combined methods render good results. Some authors have tried the malaria treatment. Autohemotherapy has been tried by Dr. Burak in several dozen cases, but has led to no improvement. Conservative methods are not very useful. Surgical treatment gives rapid and lasting effects; stenosal scars later detract from the excellent results. For operation on the larynx, trachea and bronchial tubes, laryngotracheotomy is used. If one adapts oneself to the individual case and combines operative and conservative methods (such as dilatation, roentgen therapy vaccinochemoproteinotherapy and diathermy), one can obtain surprising results in cases considered as despaired of. It is believed that the operative methods are the most efficacious.

Serology and Allergy of Scleroma. Dr. Eduard Neuber, Debrecen, Hungary.

Dr. Neuber reviewed briefly early active and passive immunization and the immunity reaction, now possessing only historical value. He believes that the following considerations encompass and exhaust the questions pertaining to the serologic reactions in scleroma.

1. Whether specific protective materials occur in the serum of scleromatous patients or in animals immunized with scleroma-like organisms, and if so, at what time, preferably, they are present in such a number that they can be demonstrated by serologic reactions.

2. Whether organisms of the encapsulated bacteria group (such as Friedländer's bacillus, Frisch's bacillus, Abel-Löwenberg's bacillus, Bacillus lacticus and Bacillus aerogenes) can be differentiated by means of serologic reactions.

3. All technical questions concerning recovery of antigens and treatment with immune serum.

He discussed the complement-fixation, agglutination and precipitation tests with these considerations in mind and with special attention to the latest achievements in serology.

Much can be accomplished in the detection of hidden cases of scleroma in endemic regions by the use of serologic tests, in particular complement-fixation tests. On the basis of his own investigations the author maintained that immune biologic reactions and vaccinotherapy will be the most successful methods of treatment in the future.

He laid great stress on the allergic reactions in differential diagnosis. Specific allergic reactions are obtained by intracutaneous methods (Mantoux-Roux). In most cases the allergic reactions are differentiated within the encapsulated bacteria group and are consequently type-specific.

EPIDEMIOLOGY OF SCLEROMA. PROF. DR. BELINOFF, Sofia, Bulgaria.

The contagiousness of scleroma remains a matter for debate. Authorities are divided on the subject. Putschkowski and Barraud consider the disease contagious, whereas Streit, Hajek, Šercer and Szmurlo do not. Since 1918, rhinoscleroma has been known as a contagious disease. The Ukraine and White Russia have been the foci of greatest incidence. Czecho-Slovakia (262 cases) was the first country to establish a special commission for the campaign against scleroma. Poland formed a committee in 1931 and has a considerable number of cases (798) on record. Yugoslavia has a record of 136 cases; Germany, 57; Rumania, 26; Hungary, 23; Italy, 61; Dutch East India, 26 (up to 1931); the French colonies, 8 (unknown to native French people); Switzerland, 8 (Canton du Valais); Lithuania, 6; Bulgaria, 5; Turkey, 2; Denmark, 1; the United States, 47 (4 patients were native Americans), and Central America, 3.

Scleroma is more prevalent among women than among men. It commonly occurs between the ages of 15 and 35. Scleromatous patients are 94.5 per cent laborers and peasants, 4.2 per cent white clerical workers and 1.3 per cent intellectual workers. The disease is localized mainly in the nose and larynx. Galicians have been the most frequent agents in the spread of scleroma. Many Bulgarian merchants and laborers going into Russia, Rumania, Poland and Czecho-Slovakia have acquired the disease, but not one has transmitted it to his family. The disease has increased in Russia and diminished in Germany. The increase in Russia is credited to the conditions of poverty there.

A CASE OF RHINOSCLEROMA. DR. NUÑEZ, Spain.

LOCALIZATION OF FRISCH'S BACILLUS AND IMMUNIZATION. DR. RENATO SEGRE, Torino, Italy.

IMPORTANCE OF SEROLOGIC ACTION IN THE DIAGNOSIS OF SCLEROMA. DR. ARNALDO MALAN, Torino, Italy.

INTRODUCTION TO THE STUDY OF SCLEROMA IN COLOMBIA. DR. A. BRIGARD, Bogota, Colombia.

DISCUSSION

DR. STREIT: It is not necessary for a disease to have two names. It is often confusing. This condition should be called "scleroma" and the term "rhinoscleroma" should be forgotten.

DR. NEUMANN: A characteristic of scleroma is that it occurs without ulceration. The organ becomes smaller through shrinking without loss of substance. This may be compared to the shrinking of a shoe sole which is first soaked in water and then brought close to a fire. There is a characteristic unpleasant, sweetish odor. The disease appears in different places and in different forms.

While infiltration is still at the surface, shrinking may occur at the posterior nares or at the posterior surface of the soft palate.

DR. BELINOFF: Dr. Brigard's communication shows that scleroma can present different characteristics. The destructive form of scleroma is rare in Europe; in Colombia, however, this form is frequently observed.

DR. STUPKA: The rare cases of scleroma with erosions and ulcerations are mainly due to secondary infections. This is substantiated by the research of an authority on scleroma, Dr. Pienacek of Krakow, Poland.

PROPOSITIONS ON SCLEROMA ADOPTED BY THE CONGRESS.

Dr. Streit's proposal of the name "scleroma" in place of "rhinoscleroma" and "pharyngoscleroma" was unanimously accepted by the congress.

The Second International Congress of Otorhinolaryngology, at Madrid, Sept. 29, 1932, made the following resolutions:

1. A permanent international committee for the study of scleroma shall be elected, composed of members from Russia, Poland, Czecho-Slovakia, Yugoslavia, Rumania, Bulgaria, Germany, Switzerland, Austria and South America.

2. This committee will distribute projects to be worked out in the various countries, and in four years it will present its evidence at a third international congress on otorhinolaryngology.

3. The committee will edit a special publication called the *Bulletin*.

4. An institute of study will be founded for continuous systematic research on and propaganda against the disease. The organization of this institute will be financially dependent on the individual governments (unanimously approved).

ENDOSCOPIC TREATMENT OF SUPPURATIVE DISEASES OF THE BRONCHI AND LUNGS. DR. CHEVALIER JACKSON, Philadelphia.

Fundamental to all study and all treatment of suppurative disease of the lungs is the recognition of bronchial obstruction as an etiologic factor. Whether the attacking organisms are anaerobic or not, free ventilation and drainage are essential to the highest efficiency of the defensive powers of the lungs in all bronchial and pulmonary diseases, with the possible exception of tuberculosis.

The bronchoscope affords the only means of determining positively the character of the obstruction in a particular case; usually it affords the only practical means of removing the obstruction.

One cause of suppurative disease of the bronchi and lungs is obstruction by a benign tumor. The most common of all growths is a tumor-like formation which on histologic examination shows only chronic inflammatory tissue. This does not mean chronic mucosal bronchitis; there is no true mucosa covering many of these tumors. Why the chronic inflammatory tissue should take on a rounded tumor-like form is an interesting question. It seems possible that the respiratory elongation and shortening of the bronchi twenty or more times a minute, day in and day out, may have a pill-rolling effect on the slowly accumulating mass of newly formed inflammatory tissue.

The suppuration may be the result of (1) the breaking down of the growth or, more often and earlier, (2) obstruction to ventilation and drainage which causes first atelectasis, then drowned lung and then abscess. Endobronchial growths may be removed if they are in the early stages and small. Implantation of radon is an effective method of treatment and affords a target for radiation. In most cases high voltage roentgen therapy arrests malignant growths for a time; in a few cases the effect is permanent. If bronchoscopic examination were made in every case of nontuberculous cough in many of these patients the malignant disease would never reach the suppurative stage. In some clinics, as an average, approximately 200 cases of malignant pulmonary disease are diagnosed annually by bronchoscopic biopsy.

Diagnostic bronchoscopy is required to determine whether or not bronchoscopic treatment is indicated in a particular case. It might be added that diagnostic bronchoscopy in a modern well equipped medical and surgical clinic for thoracic diseases constitutes one of the important parts of the preliminary study of every case. Only certain cases are found suitable for bronchoscopic treatment.

The bronchoscope bears the same relation to intrathoracic disease as the vaginal speculum does to pelvic disease. Specular examination anywhere (1) may be negative, (2) may demonstrate a condition amenable to treatment through the speculum, (3) may reveal a condition amenable to treatment by radiation or physical therapy or (4) may show a condition that should be dealt with by external surgical intervention.

Bronchoscopic aspiration often checks the progress of pulmonary abscess toward the pleura, but in cases of acute peripheral abscess with pleural involvement bronchoscopy is so rarely beneficial as to be contraindicated.

Asthma, when suppurative, is usually benefited by bronchoscopic aspiration. In the earlier stages of allergic asthma much of the patient's distress comes from bronchial obstruction (check valve, by-pass valve or stop valve types) by a thick, viscid secretion that the patient cannot expel. Great relief, temporary or permanent, comes from bronchoscopic aspiration of this secretion; it is a prophylactic measure against suppurative asthmatic bronchitis.

In the stage of impending asphyxia, marked relief and a turn of the tide may be obtained by bronchoscopic aspiration. Almost every patient with any indication of drifting into suppurative "unresolved pneumonia" should have bronchoscopic aspirations. Almost always a prompt arrest of a postpneumonic suppurative process may be expected from the addition of this form of treatment. Postoperative pneumonia, when not embolic, is usually secondary to postoperative atelectasis; pneumonia as well as the formation of abscesses is preventable by bronchoscopic aspiration in the primary stage of atelectasis. In its incipience lobar pneumonia can be arrested by bronchoscopic aspiration. According to Coryllos and Birnbaum, pneumonia, in some cases at least, is a pneumococcic obstructive atelectasis relievable by bronchoscopic aspiration. In many cases it is possible to arrest bronchopneumonia by the addition of bronchoscopic aspiration to the general care and management. In some cases the effect is strikingly prompt. In almost all cases a turn for the better is noted within twenty-four hours.

Bronchoscopic observations at the bronchoscopic clinic indicate that: (1) The purulent secretions when first produced in bronchiectatic bronchi are very thick and of such great viscosity that they cannot be expelled under the existing impaired conditions of expulsion; (2) Nature's way of aiding expulsion is by the breaking up of the viscid secretions into more fluid and thus more easily expelled secretions; (3) this change in the character of the secretions is brought about by bacterial activities, chiefly saprophytic, but other agencies may contribute; (4) the products and by-products of these changes are intensely irritating to the bronchial walls and are one of the factors in the changes in those walls as well as in the perpetuation of the disease. Though this use of the tracheobronchial tree as a septic tank for alteration of the character of the contents may enable the person ultimately to get rid of the pus by lessening its viscosity, the resultant structural changes in the bronchial walls, and especially the destruction of the cilia, constitute a tremendous price to pay for riddance of pus. By way of contrast, it may be parenthetically stated that by bronchoscopic aspiration one removes the freshly generated thick pus of high viscosity without waiting for it to reach a lower degree of viscosity by rotting.

Bronchoscopic aspiration is supplemented by the "trussive squeeze." That is, the compression of the lung during coughing squeezes the pus out of the parenchyma and the smallest bronchi to within reach of the aspiration tube. For satisfactory bacteriologic study as well as for the preparation of the most useful vaccines it is essential for the bronchoscopist first to clean out the bronchial septic tank and then, in a separate collector, to remove the residual pus, usually in small amount, directly from the suppurative focus.

Bronchoscopic aspirations, if instituted early, restore the defensive powers of the lungs by (1) taking the load off the overwhelmed cilia, (2) restoring ventilation and drainage, (3) forestalling stagnation and (4) permitting early discovery and removal of bronchial obstruction. By arresting the suppuration in the acute or the subacute stage, bronchoscopic aspirations will prevent the development of bronchitis, bronchiectasis, the bronchiectatic septic tank, fibrosis, adhesions and other extensive structural changes in the lung which, once established, are the largest factor in the perpetuation of chronic suppuration and its sequelae.

In order that this life-saving work may be developed to the utmost, two things are necessary.

1. There should be eternal vigilance to detect in the earliest stages those mild, slightly suppurative conditions that linger on, insidiously and progressively growing worse, after acute conditions such as measles, pertussis, bronchopneumonia, pneumonia, atelectasis, diphtheria, tonsillitis, influenza and especially the acute respiratory infections often called a "cold." In all such cases in which prompt cure does not follow from medical care, treatment of the nasal accessory sinuses and diagnostic bronchoscopy should be done at once. In rare instances a foreign body may be found, and more often plugs of secretion, crusts, swollen bronchial orifices, benign tumors, tumor-like inflammatory formations, granulations, granulomas or some other form of bronchial obstruction. In any case prompt relief can be afforded by bronchoscopic treatment. Often the condition is an incipient chronic suppuration that will clear up promptly on the addition to the previously unsuccessful medical care of weekly or biweekly bronchoscopic aspirations together with the administration of vaccines prepared from the uncontaminated material bronchoscopically aspirated from the suppurative focus in the tracheobronchial tree, after aspiration of the superjacent saprophytic pus.

2. Every large hospital should have a well equipped bronchoscopic department with a well organized staff prepared at all times to make diagnostic and therapeutic bronchoscopies in children without general anesthesia. In case of bronchoscopy to detect a foreign body, which is done perhaps only once in a patient's lifetime, general anesthesia is permissible if the bronchoscopist desires to use it; but in case of bronchoscopic aspiration for suppurative conditions, repeated perhaps twice weekly for a number of weeks or months, the use of general anesthesia would prevent the unhesitating resort to this method that is essential for its greatest usefulness. Thousands of consecutive bronchoscopies performed without resorting to general anesthesia have demonstrated it to be unnecessary.

In suppurative disease of the bronchi and lungs the main need is to improve ventilation and drainage, just as in suppurative disease of the nasal accessory sinuses. As in the latter, the preferable route of drainage is through the natural passages when possible, but it is not always possible.

Collective statistics show that, in 43,710 endoscopies performed by 190 bronchoscopists, the mortality was less than 0.1 per cent, and that if one considers the bronchoscopy apart from the condition for which it was done, there was no mortality. That is, no death occurred from the mere insertion of the bronchoscope. The course of the disease was influenced favorably in 76 per cent of the cases of acute pulmonary abscess, in 52 per cent of the cases of chronic pulmonary abscess, and in 80 per cent of the cases of bronchiectasis. Of the patients with bronchiectasis, only those with early unilateral cases were cured; in bilateral, long-standing cases the malady was considered favorably influenced when the patient gained weight, improved in morale or was restored to economic usefulness, or when the odor was ameliorated so that the patient was no longer an outcast.

In pulmonary diseases caused by other factors than foreign bodies bronchoscopy is not a substitute for other methods of diagnosis and treatment.

The greatest future advances in the knowledge of every phase of bronchopulmonary diseases and their treatment will come from the use of the bronchoscope as an aid to laboratory and clinical research in the living bronchi and lungs.

ENDOSCOPIC TREATMENT OF INFLAMMATORY LESIONS OF THE ESOPHAGUS DUE TO FOREIGN BODIES. DR. C. O. VON EICKEN, Berlin, Germany.

Foreign bodies in the esophagus are much less dangerous than formerly. As a sequel to the progressive development of the endoscopic technic, it has become rare to observe perforating lesions following the extraction of a foreign body. The great dangers of mediastinitis as well as those of mediastinotomy were completely avoided by Seiffert, thanks to endoscopic surgery. Since emphysema is considered the surest symptom, the hospitals in Berlin have developed the habit of using roentgen examination in all suspected cases. This helps to reveal the accumulation of air. Since the development of the disease depends eventually on the type and the extent of the perforation, the indication for the operation must be special for each case. In some cases perforations heal spontaneously. Abscesses of periesophagitis must be operated on. The lives of numerous people have been saved by the combined external and internal operation of Seiffert.

MY METHOD OF PLASTIC OPERATION ON THE ESOPHAGUS. DR. J. LASAGNA, Parma, Italy.

The causes of esophageal stenosis are foreign bodies, outer pressure and alterations of the esophageal wall. The most frequent causes of stenosis are tumors, caustic fluids and hyperplasia of the muscles and connective tissue.

These stenoses frequently are situated at the beginning and at the lower extremity of the esophagus, and they have a bad functional prognosis because they persist and gradually cause complete closure of the esophagus. For this reason it is necessary to correct the caliber of the esophagus. To accomplish this, operative and nonoperative means have been tried; the surgeon and the specialist have especially sought success with plastic operation.

König and Homayer were the first to apply successfully a piece of aponeurosis to the lesions of the mucosa.

Kostendo and Ronbaceff have made three types of experiments. In the first group they used a free piece of aponeurosis; in the second group, after having excised a part of the esophagus they made a suture of the two extremities and superimposed a piece of aponeurosis; in the third group, they substituted a piece of aponeurosis. They did not obtain good results.

Nasetti made experiments on the mucosa, the musculature and the entire wall of the cervical portion of the esophagus, using grafts of aponeurosis from the neck and leg. He observed degeneration and disappearance of tissue in certain cases and cicatricial stenosis in other cases.

Razzaboni performed plastic operations with peritoneum, aponeurosis and muscular fibers of the abdomen on the cervical portion of the esophagus and did not obtain good functional results. In a second series of experiments he used part of the intestine to replace a piece of the cervical portion of the esophagus.

As these experiments had not shown good results, another plastic operation that would employ more vital flaps and more pedicles was desired. Muscles of the neck were chosen which would cause no alteration of the motility of the bone and cartilage and which might be of satisfactory length and closer to the esophagus, that is, the sternothyroid and sternohyoid muscles.

Dogs were used in the experiments. The operative technic was as follows: (1) Preliminary anesthesia was secured with morphine; (2) an incision was made in the middle line of the skin and superficial tissue of the neck of the hyoid bone at the sternum; (3) the sternohyoid and the sternothyroid muscles on the left were isolated; together with the intermediate aponeurosis which unites the two muscles, they were cut near the superior insertion and were then turned down; (4) the nerves and blood tissues were brought to the exterior, the thyroid gland being on the right; the esophagus was separated from the trachea and from other parts, and two sounds were passed below; (5) the section of the esophageal wall which was to be replaced by the muscle graft was cut, and the walls were held drawn; (6) the muscles were brought to the esophagus, and a piece of the muscle,

much larger than the raised flap, was sutured with catgut, beginning from below; (7) the muscles and skin were sutured. The experiment was performed on six dogs, five of which showed successful results. In some cases a hemicircumference was raised; in others, the complete circumference of the esophagus. In the former cases the muscles on the left were used; in the latter, those on both sides.

The dogs were not fed for forty-eight hours. They were fed for a month or more with fluids and were killed from two months to a year after operation. The dogs were examined roentgenologically many times; at necropsy the muscles and the caliber of the esophagus were examined. The dogs gained weight, and the esophagus preserved its caliber and form.

Histologic examination revealed that the mucosa was formed of from three to four layers of cells, the superficial cubic and the flat inferior cells.

The submucous tissue was normal; the glands were well developed and numerous; the muscular tissue of the flap was normal, and there were numerous vessels with connective fibers that united the esophagus with the graft.

In conclusion, the plastic muscular operation is the best one for stenosis of the first portion of the esophagus. Good results have been obtained, depending on the vitality of the chosen tissue in situ, on the reconstruction of the mucosa with its epithelium and glands and on the absence of cicatricial connective tissue.

This method may be applied in cases of clinical stenosis in order to aid in restoring the altered and lost function of the cervical portion of the esophagus.

DISCUSSION

Dr. Faire: In pulmonary abscesses, one must consider the reaction of the surrounding pulmonary tissue. A type of pulmonary abscess occurs for which no treatment has been described and which shows a precise therapeutic orientation. This type of abscess has fibrous bundles and is cavitary; contact of the tube with the walls of the cavity shows an intense fibrous resistance. The surgical treatment consists in external drainage. In those seats of suppuration which are easiest to clean by bronchoscopic absorption, neither granulation nor collapse is produced.

In regard to bronchoscopic treatment of interlobar abscesses, Dr. Jackson points out the great danger of attempting to establish the drainage opening with a tube. Yet other operations are dangerous in that respect, and many patients die during operation because of acute pneumothorax. Interlobar abscesses on the left side are particularly suitable for treatment by bronchial aspiration.

Dr. A. Soulas (for Dr. Chevalier Jackson): Bronchial obstruction exists in all cases of bronchopulmonary suppuration. It is rarely complete. The relief of the bronchial obstruction is an endoscopic maneuver of the first order. In rare cases the obstruction causes emphysema or atelectasis. This is seen especially in cases of foreign bodies and of postoperative pulmonary atelectasis.

According to Professor von Eicken there are many cases in which secretions no longer indicate atelectasis but represent an inundation caused by a type of cupping-glass suction. The obstruction causes not atelectasis but a drowned lung. These inundations occur rapidly and are cured rapidly.

Posttonsillectomy abscesses are rare in France. Pulmonary suppurations respond to bronchoscopic treatment whether they are multiple or generalized. But if the abscess is situated high, surgical intervention is preferred to endoscopic treatment.

Ethmoido-Orbital Epitheliomas: Combined Treatment with Radium and Surgical Intervention; Late Results. Dr. A. Hautant and Dr. O. Monod, Paris, France.

Epitheliomas of the ethmoido-orbital region are most often prismatic; sometimes they are epidermoid, or a combination of epidermoid and prismatic; sometimes they are colloid. Their radiosensitivity is attenuated. They progress very slowly. Metastases are exceptional. The danger from these neoplasms lies in their propa-

gation to the meninges and the ensuing infection of the endocranium. It is always important that they be removed. The operation must be conducted very meticulously. Skill must be exerted to extirpate the growths completely, cutting around them into the healthy tissue as far as possible.

Curettage and morcellation should be avoided.

The operation should be completed immediately by an intracavitary application of radium distributed among ten or twelve fields. A dose of 20 disintegrated millicuries would be distributed in four or five days without extending beyond its goal.

This technic, applied in twenty-one cases during a period of four years, brought favorable results from four to twelve years after treatment in 38 per cent of the cases.

TECHNIC OF THE USE OF RADIUM AND ROENTGEN RAYS. DR. LEDOUX and DR. SLUYS, Bruxelles, Belgium.

Dr. Ledoux and Dr. Sluys studied the elements of radiology and roentgenology and the methods of application.

They presented some general considerations on the histologic forms found in the field of otorhinolaryngology, on neoplastic extension and on other local factors which serve as a guide in the choice of the most reasonable methods for use in each localization.

TREATMENT OF CANCER OF THE TONSILS. DR. FERNAND LEMAITRE, Paris, France.

Operations for cancer of the tonsils are always difficult, mutilating, dangerous and only fairly successful.

Radiotherapy, on the contrary, is easily applied, seldom mutilating and encouraging. Three cases were observed. In case 1, the cancer did not present adenopathy, at least not clinically. Radium was applied to the tonsil and then roentgen radiation to the lymphatic tributary zone. Radium was applied by means of several pointed needles, some inserted vertically or obliquely in the tonsils and the pillars, the others horizontally in the velum at a level with the superior pole of the tonsil and in the tongue close to the anterior pillar.

The dosage varied from 10 to 20 millicuries, and the needles remained in place from five to ten days. This dose, according to the degree of extension of the epithelioma, is stronger in recent than in old processes; it is well tolerated and seems more efficacious.

The author had never seen hemorrhages following or during radium puncture. The cases described seem to have been due to the resorption of the epithelial tissue which had already invaded the tonsillar vessels.

Roentgen radiation was applied afterward. Generally two fields from 10 to 12 cm. by from 16 to 20 cm. permit the application on each field of 25 Hampson units in six or seven hours, in the space of from ten to twelve days.

In case 2, the cancer was accompanied by adenopathy. The treatment of the epithelial ulceration was the same as in the preceding case: radium and roentgen radiation. But surgical intervention also was necessary. Though small and mobile adenopathies can retrocede under the action of the rays, this does not hold true for large adenopathies; they require surgical intervention. The operation consisted of a complete curetting of the neck involving all of the carotid region, even the subclavicular cavity and the submaxillary cavity, with exeresis of the sternocleidomastoid muscle and the internal jugular vein. It is preferable to begin with radium puncture of the tonsils, to continue with cervical roentgen therapy and to end with surgical operation. Yet the operation may be performed during the application of the rays, which are generally applied twice, before and after the operation.

Case 3 was one of the type in which there is profound carcinomatous infiltration of the muscular planes (trismus) or the vessels (important spontaneous hemor-

rhages) or a recurrence despite a reasonable and even maximum application of radiation. Surgical intervention is necessary in these cases.

The epithelial ulceration of the tonsil which responds so well to radiation does not seem relieved by operation. In cases of small and voluminous adenopathies which do not yield to roentgen therapy, surgical treatment is necessary.

Without surgical treatment in certain cases there would be no response to palliative treatment. The treatment of epitheliomas, owing to radium and the roentgen rays, has often been successful. Unfortunately little can be said of certain tumors of the tonsils which remain delusive.

Lymphosarcomas, because of their extreme radiosensitivity, disappear rapidly under the action of radiation but soon recur. They may disappear from the tonsils but progress in all of the cervical regions. This holds for tonsillar lymphocytomas likewise, especially in young patients.

Lymphoblastomas are as radiosensitive as lymphocytomas, and since they cannot be accompanied by adenopathy the rapid resorption of the tumor cannot be followed by distant metastases, ganglionic or otherwise. In two cases lymphoblastomas of the oral cavity were cured with the greatest ease by roentgen therapy. Typical lymphosarcomas, like reticulomas, are considered very radiosensitive.

Lympho-epitheliomas, which are histologically epitheliomas, biologically belong to the lymphosarcomas; they are radiosensitive, and when situated in the oral cavity they give rise to profound adenopathies.

All in all little seems to be known of the cancerous tumors of the tonsils (lymphosarcomas, reticulo-endotheliomas and lympho-epitheliomas). Their radiosensitivity is great, they are often accompanied by adenopathies and they disappear rapidly, either spontaneously or following the action of the radiation. They do not respond to surgical intervention and their prognosis is generally unfavorable, except for those unaccompanied by adenopathies, which are benefited greatly by radium therapy. The study of different methods of application of radiation will perhaps aid in solving this problem. In all cases of epithelioma, radium and roentgen therapy have given excellent results.

THE TECHNIC OF ROENTGEN TREATMENT OF SPINOCELLULAR EPITHELIOMAS OF THE LARYNX THROUGH ENDOLARYNGEAL FOCI. DR. GAULT, Dijon, France.

TREATMENT OF MALIGNANT TUMORS IN OTORHINOLARYNGOLOGY WITH ONCO-VACCINOTHERAPY. DR. CITELLI, Italy.

DISCUSSION

PROF. A. G. TAPIA: The ideal treatment of cancer of the larynx would be a process curing the disease without mutilating the organ, as most operations do.

When one realizes that the cancer cells disappear after radium and roentgen treatment to such a degree that the vocal cords reacquire their nacreous color and normal mobility, one cannot help but think that physicians are not far from developing a radical cure for this terrible disease. Twelve years ago a cure resulting from radiotherapy was exceptional. Today good results are more frequent. Despite all the efforts of learned investigators cancer remains an enigma. It is said that most cancers of the larynx are prickle cell and basal cell types which are unusually radiosensitive, and consequently in those cases the effects of the radiation are more evident. But in proportion to other types of cancer they are no more curable; some growths respond immediately to treatment and never recur, but others recur immediately after treatment.

Cancer is a very complex disease about the pathogenic evolution of which little is known and in which there are perhaps biochemical and constitutional factors as yet unknown.

Of all methods of treatment, surgical intervention must still dominate. If one is to choose between a laryngofissure and a hemilaryngectomy, one must choose

the latter. Young patients, from 25 to 35 years of age, must be radically operated on.

Radiation should be applied: (1) in cases in which patients conscious of the value of each method refuse surgical intervention; (2) as an auxiliary to surgical treatment; (3) in cases in which intervention is contraindicated (serious lesions of the heart, pulmonary tuberculosis), and (4) in cases in which operation is technically impossible.

PROFESSOR VAN DEN WILDENBERG: It is generally accepted that surgical operation is the most efficacious therapy for cancer.

When the cancer is largely irradiated thyroidectomy can be performed without the least inconvenience. I have successfully performed thyroidectomies and laryngectomies after irradiation. This, however, was when radiotherapy and roentgenotherapy were in good standing; otolaryngologists now condemn the use of radiation before operation.

Partial and total laryngectomy are infinitely more efficacious than teleroentgenotherapy.

In the treatment of endolaryngeal cancers applications of physical agents are advisable as a consolidation treatment after total laryngectomy in certain cases. The eventual beneficial action of radiation is not injurious.

DR. MAYER: Radiation treatment of laryngeal carcinoma is still experimental. One consequently may use radiation only in cases in which operation is impossible and in cases in which operation is refused. Patients must be told how insecure the results of radiation are. It is held that in some cases, although radiation was applied, recovery might have occurred without radiation.

DR. REBATTU: The more reliable authors make a histologic classification of tumors so as to permit a precise record of the nature of the tumor and the indications for surgical intervention, radiotherapy and roentgen therapy. This is of great necessity in making the choice of treatment. The best results that I obtained in velotonsillar cancer were in a patient with spinocellular epithelioma, histologically very malignant, with carotid adenopathy.

Two patients with lymphoblastic sarcoma of the tonsils were submitted to the same radiation treatment, to which they responded differently. In one, a woman aged 20, the tumor disappeared rapidly and recurred in less than two months; radiation was again applied; death occurred four months after the onset. The second patient, a woman, aged 75, with cardiac disease, who was treated in the same way, had a recurrence eight months after treatment which disappeared after the second irradiation; she died six months later of heart failure. Thus, when a tumor seems radiosensitive, it is impossible to know whether the radiation (roentgen or gamma rays) will produce lasting results or whether there will be recurrences.

DR. PROBY: My associates and I use the intrabuccal tube for easier application of radium in the treatment of cancer of the tonsil. If the cervical route is used, radiotherapy is begun after excision of the cutaneous flap, which should theoretically stop the radiation.

We have not obtained better results with this technic than with regular radiotherapy. The apparatus used in radium treatment consists of a hook attached to the last upper molar supporting a stem to which is attached a network of silk thread forming a hammock to hold the radium tubes.

DR. CASADESÚS: Surgical intervention must be recommended before the application of radiation in all cases in which the tumor is operable. The majority of patients improve after roentgen therapy, but ordinarily recurrences are very brusque and prevent further operation. In cases in which the tumor is circumscribed, I do not give postoperative radiation; this is reserved for cases in which the growth is exceptionally extended and the ganglions are invaded.

PROFESSOR VON EICKEN: Otolaryngologists should meet every four years to collect the various experiences with operation and radium therapy and determine the respective applications of the two methods.

The tumors confined to the vocal cords should be treated with radium, especially if small. In from three to four weeks the tumor usually disappears. In certain cases following any infection, e. g., influenza, perichondritis occurs from one to two years after treatment, thereby requiring operation. If a prolonged disease of the vocal cords is not successfully treated with radium, there always remains the possibility of surgical removal of the diseased tissue. Both radium and roentgen therapy are to be recommended in place of surgical intervention for multiple tumors of the hypopharynx. In order to open the pharynx a large external incision is made in the sternocleidomastoid muscle, followed by a division of the homohyoid muscle, after which the carcinomatous region is exposed in the shape of a pocket. In this pocket the radium containers, wrapped in rubber, are placed for a specified time and then removed. It is astounding to see how the multiple tumors disappear and the function of swallowing becomes normal.

In tumors of the epipharynx the best results are obtained with roentgen and radium therapy.

Dr. Faire: Small localized epithelial tumors are often so aggravated by radiation that death results. Sometimes the tumor disappears but asthenia ensues, progressing to cachexia, which kills the patient with or without (or with few) manifestations of reaction. In other cases the tumor grows and becomes necrotic after irradiation. The primary anabolistic and catabolic manifestation is in relation with the irradiated lipoid tumors.

Dr. Quix: I do not agree wth Professor von Eicken that the functional result after radiotherapy for a small tumor of the vocal cords is always better than the functional result after laryngofissure.

Many patients who have been operated on for laryngofissure speak normally after operation and continue to exercise their professions (e. g., as judges or priests). One priest after hemilaryngectomy sang at the mass in such a way that one would never know he had been operated on.

Dr. Stupka: O'Dryer's intubation tube has been successfully applied in carcinoma of the larynx. It is recommended for irradiation after thyrotomy and in cases in which a general contraindication against operation exists.

Dr. St. Clair Thomson: In radiotherapy for intrinsic cancer, the importance of preserving a normal voice is exaggerated, considering that the majority of patients are 60 years or over.

A cure lasting from ten to twenty years may be obtained with laryngofissure, 80 per cent of patients retaining their normal voices.

With the progress of early diagnosis more patients will be cured through laryngofissure without the inconvenience of radium.

Dr. Louis Ducuing: The Anticancer Center of Toulouse treated 200 patients with laryngeal cancer. It was found that radium treatment of intrinsic cancer of the larynx does not give good results. Radiotherapy according to Contano's technic, in fractional doses, gives excellent results.

In the majority of cases one of the following surgical operations is necessary: (1) ablation of the tumor followed by radiotherapy for circumscribed cancers of the vocal cords; (2) frontal or Hautant's hemilaryngectomy for extensive cancers; (3) total laryngectomy according to the Glück-Tapia process for extensive cancers without laryngeal confinement.

Dr. Gil y Gil: During 1932, 58 patients with laryngeal cancer were treated only with roentgen radiation at the National Institute of Cancer in Madrid. The dose was given at two day intervals, and on the fourth or sixth day saturation doses according to Pfahler were given. In several patients edema of the glottis developed following treatment, and an emergency tracheotomy had to be performed. The results in the long run were not favorable. To avoid complications of asphyxia, fractional doses were used for some patients. One hundred and fifty roentgens were first given; on the following days, from 200 to 300 roentgens. In general, only lateral fields were used. The superficial dose was from 1.8 to 2.5

roentgens per field. The full dose was from 3.5 to 4 roentgens. The treatment was given daily for three weeks. The results were favorable.

Dr. Taptas: For partial or total removal of the larynx surgical intervention is the best treatment. In a certain patient total laryngectomy was performed, the epiglottis being preserved. Recurrence of the cancer occurred one year later. The hyoid bone had to be raised with two thirds of the tongue and a large part of the remaining portion of the larynx. The patient was alive ten and one-half years after operation.

After radium treatment, recurrence or necrosis of the cartilage may be the result. In recurrence the diminished vitality of the tissues makes operation virtually impossible.

Professor Bilancioni: The universities of Pisa and Rome follow the eclectic school. A wide variety of operations are performed, always followed by applications of radium.

Professor Torrigiani: Standardization of treatment is not possible. Some patients respond readily to irradiation and others to surgical intervention. Fundamentally, operation is the better choice.

Unusual Results in Chronic Suppurative Otitis Media with the Iodine Powder Treatment of Sulzberg. Dr. M. D. Lederman, New York.

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Myiasis in the Bony Cells of the Mastoid with Reflex Respiratory Paralysis. Dr. Jimenez Cervantes, Madrid, Spain.

Book Reviews

The Principles and Practice of Otology. By F. W. Watkyn-Thomas and A. Lowndes Yates. Price, $8.25. Pp. 555, with 199 illustrations. New York: William Wood & Company, 1933.

This is a volume of 555 pages with 199 illustrations, 5 being in colors. The authors state that their aim is to combine the full details of practice, to help the general practitioner-surgeon who treats his own patients with diseases of the ear, with enough discussion of principles to make the book an introduction for the intending specialist. A book such as this, written by two authors, has evident disadvantages. One is the extending of the length of the text with unavoidable repetitions. The authors have adopted a style quite their own in presenting the subject, a style which is different from the formula ordinarily adopted in preparing a textbook. Many of the illustrations are sketchy and poorly drawn. On the other hand, the illustrations include a number adapted from other texts; on the whole, the volume is rather profusely illustrated.

There is a good deal of space given to a discussion of the physiology of the function of hearing, and in this discussion emphasis is placed on questions which ordinarily are not included in a presentation of the subject. Emphasis is laid on the fact that a defect for hearing the voice is not always proportional to the degree of deafness ascertained in separate parts of the tone range; also, that many persons suffering from defects in hearing have compensated for this defect. This compensation is, after all, a matter of mental alertness and intelligence and has nothing to do with the type of defect.

The authors express doubt as to whether the Rinne, Schwabach and Weber tests as ordinarily carried out give sufficient data to recognize the type, the degree and the curability of the patient's deafness. As a matter of fact, testing the hearing for the voice is the only way one has of determining the actual handicap which a patient has from his defect in hearing, and a defect more or less extensive in the upper tone range does not necessarily carry with it the degree of handicap which goes with a lesser defect in the lower tone ranges. Next in importance to determining the actual handicap from the defect in hearing for the voice is the problem of determining the location of the pathologic condition causing the deafness, whether this is due to conditions which interefere with the conduction of sound impulses to the internal ear or whether it is the result of alterations in the nervous mechanism itself. Methods for determining these facts are well established and include the intelligent application of the Rinne, Schwabach and Weber tests, together with an evaluation of the extent of the defect in the various parts of the tone range. These tests have become a fundamental part of the study ot an otologic case and are not at all displaced by any of the newer tests, including tests with the audiometer.

The authors emphasize the desirability of otologists using calibrated forks. The reviewer does not recognize any very great advantage in using calibrated forks in reaching the objectives which have just been pointed out.

The authors introduce a new method of charting the hearing which to the reviewer tends to obscure rather than clarify the subject. With the introduction of the audiometer, there has developed a tendency to attempt to rely on the audiogram for supplying information as to the location of the pathologic process responsible for the defect with the expectation that the audiogram will supersede other established tests of hearing. The reviewer believes that this is putting too great a burden on the testing of the hearing with the audiometer. It has been repeatedly pointed out that the character of the chart provided by the audiogram may be exactly the same for a defect due to sound obstruction as it is when a complete study of the ear reveals a defect limited to the nervous mechanism.

The authors have stressed many conditions in diseases of the ear which, after all, are in the nature of surmises rather than established facts. For example, on page 176 one finds a paragraph captioned, "Absorption of water from mucus," which illustrates the point the reviewer is making that surmise is stated as an established fact. One could go through the entire text and point out similar examples on almost every page.

The discussion of the subject of otosclerosis brings back the confusion which was rife in the minds of early otologists concerning this frequent cause for profound deafness. When the functional tests presented the reactions typical of obstruction to sound conduction such as exists as a sequel to chronic suppurative otitis media and when a careful examination of the drum membrane and eustachian tube failed to bring to light any evidence of alterations, the older otologists, confronted with this clinical finding and without any working knowledge of autopsy observations, which have since established fixation of the stapes as the most important etiologic factor in producing this symptom complex, were left to rely on surmise as to the location and character of the pathologic condition. Some were inclined to assume that there must exist what they termed a circumscribed chronic catarrhal otitis media, placing this pathologic condition in the niche of the oval window. In the minds of most otologists, however, it was unthinkable that a chronic inflammatory process of the middle ear capable of producing the profound defect in hearing observed in chronic progressive obstructive deafness could be produced by alterations entirely restricted to the niche of the oval window and leave no evidence of a more diffuse process in alterations on the drum membrane and eustachian tube.

The term "otosclerosis" arose from the surmise that there was a diffuse atrophic process of the middle ear which left no evidence of tubotympanic disease except the fixation of the stapes. When Bezold demonstrated by autopsy observations that this particular symptom complex was not due either to a pathologic condition of the middle ear circumscribed to the niche of the oval window or to a diffuse sclerosing process involving the mucosa of the middle ear, but was dependent on a disease originating in the bony capsule of the labyrinth and resulting in bony fixation of the stapes, the misleading term "otosclerosis" which was applied to these cases was, unfortunately, retained.

The authors hark back to the period when confusion regarding the pathology of otosclerosis was prevalent. On page 218 the statement is made that "otosclerosis is undiagnosable with certainty during life." A few lines farther on the following statement appears: "In the later stages of otosclerosis there is no difficulty whatever in diagnosing the complaint for there is no condition that will produce so great a degree of deafness of this type without signs of mischief in the middle ear."

The reviewer concurs with the authors that it is only in the early stages that there may be any grounds for confusion regarding these two forms of obstructive deafness, that is, between the disease of the middle ear and primary fixation of the stapes. Primary disease of the middle ear is common in childhood and is met with rather rarely in adult life. The reviewer cannot agree with the authors that it is always necessary, in the early stages of this symptom complex, to treat each patient as having disease of the middle ear, because they state that every patient with this condition can be cured if treated early, whereas the failure to carry out early treatment carries with it the risk of sentencing the patient to lifelong deafness which might be prevented if proper early treatment is instituted. This method of reasoning is wrong, but there are still many otologists who fail to grasp the true situation. If one admits, for the sake of argument, that the early stages of otosclerosis producing incipient deafness may be confused with processes of the middle ear so mild as to be unrecognizable from alterations in the drum membrane and eustachian tube, the bald fact still remains that this incipient tubotympanic process does not carry with it the risk of producing, eventually, profound defects in hearing. Even tubotympanic processes which are easily recognized as such from typical tubotympanic findings are rarely, if ever,

responsible for those profound defects in hearing which constitute the outstanding clinical phenomena of genuine otosclerosis.

Since Bezold established the diagnosis of primary fixation of the stapes, a large number of cases have been reported in which, during life, a diagnosis of primary ankylosis of the stapes was made and in which autopsy revealed the fact that in each instance primary fixation of the stapes from disease of the bony capsule was responsible. On those who still contend that there exist circumscribed pathologic conditions of the middle ear responsible for the clinical symptom of primary fixation of the stapes, or that there exists a diffuse sclerosing process of the middle ear unrecognized by alterations in the drum membrane, rests the burden of proof, as there has, as yet, been no autopsy evidence supporting this assumption.

The authors discuss under paracusis willisiana, false, true and cross paracusis. The phenomenon of paracusis is so conspicuous that it always seems like a real phenomenon; that is, by some unexplainable factor some people with defects in hearing actually hear better in a noisy place. As a matter of fact, it has been amply demonstrated that this ability to hear better is only apparent, and it is explained by the absence of confusion created by other noises when listening to the voice, which noises are shut out because of the type of deafness, that is, alterations in hearing due to obstruction, these noises confusing a person with normal hearing. There exists no actual increase in the function of hearing in the presence of a noise. It is suspected that this explanation of the phenomenon will be found to apply to all cases of paracusis.

Atlas of Otology: Illustrating the Normal and Pathological Anatomy of the Temporal Bone. Volume II. By Albert A. Gray, M.D. Cloth. Price, 6 pounds, 6 shillings. Pp. 72, with 92 illustrations. Glasgow: Jackson, Wylie & Co., 1933.

This is the publication of a series of photographic reproductions of preparations of the internal ear. The first section deals with normal anatomy and is not particularly instructive as the photographs were made of preparations which were poorly fixed, as is usually the case with human material. The author, on the basis of these anatomic preparations, ventures a discussion of the finer structure and relations of the membrana tectoria.

The second section deals with pathologic anatomy of the labyrinth and, as such, is very welcome. The author presents, first, a series of photographs of the internal ear in a case of septic meningitis. The sections are of interest especially because of the unexpected finding of conditions which had nothing to do with the septic meningitis. One of these is the presence of a focus of otosclerosis in the otic capsule. There are preparations of the labyrinth in cases of acoustic tumor. Here, again, the most interesting findings are foci of otosclerosis. The author holds the view that otosclerosis is the result of atrophic disturbances of the organ of hearing, and it is a question whether the finding of otosclerosis in connection with acoustic tumors may not be looked on as the result of the tumors. There are other preparations showing pathologic changes found in deaf-mutism, the alterations in a case of senile deafness and changes in the nerve in otosclerosis.

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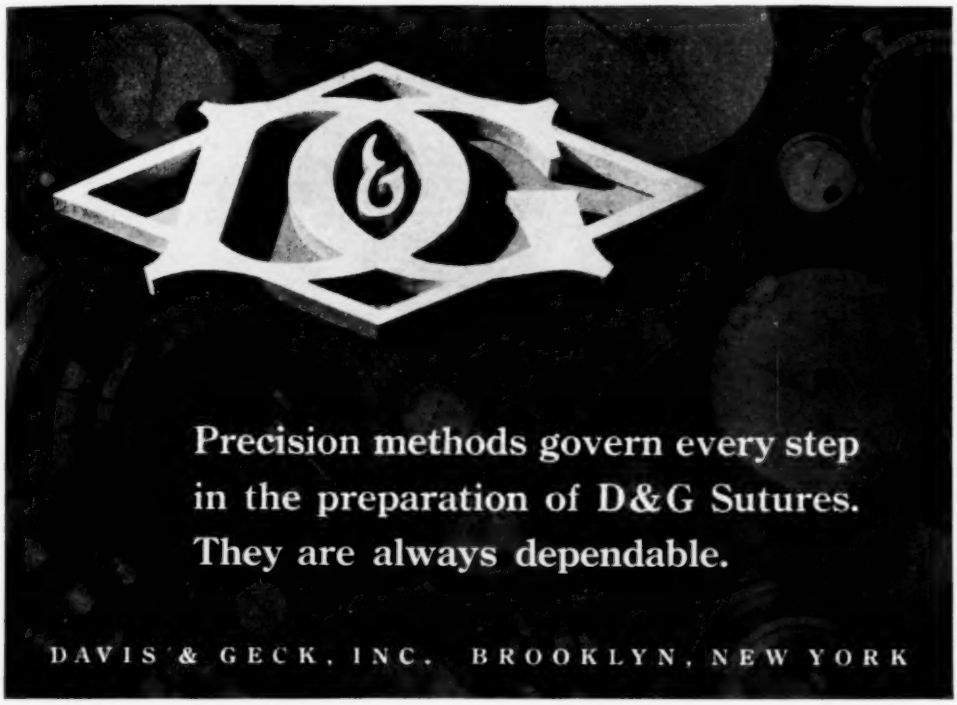
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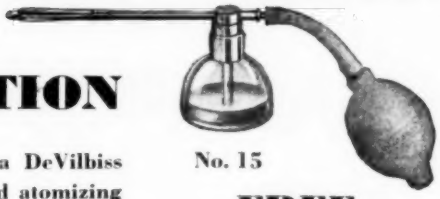

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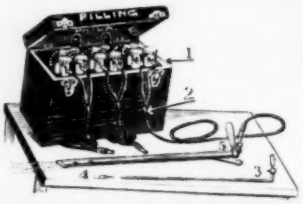

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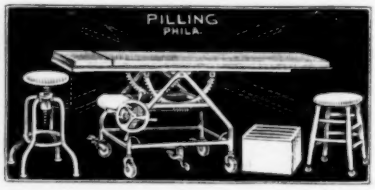

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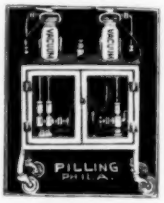

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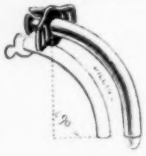

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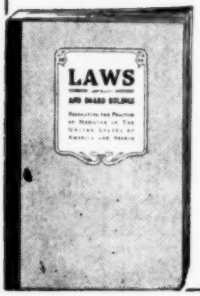

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